PREVALENCE OF GROUP B STREPTOCOCCI IN PREGNANT WOMEN AND THEIR NEONATES.

DISSERTATION SUBMITTED IN FULFILLMENT OF THE REGULATIONS FOR THE AWARD OF

M.D. OBSTETRICS AND GYNAECOLOGY

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PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

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CERTIFICATE

This to certify that the thesis entitled **PREVALENCE OF GROUP B STREPTOCOCCI IN PREGNANT WOMEN AND THEIR NEONATES** is a bonafide work of Dr. Alphy S. Puthiyidom done under my direct guidance and supervision in the Department of Obstetrics & Gynaecology, PSG Institute of Medical Science & Research, Coimbatore in fulfillment of the regulations of Tamil Nadu Dr. MGR Medical University for the award of MD degree in Obstetrics & Gynaecology

GUIDE **HOD**

PRINCIPAL

DECLARATION

I hereby declare that this dissertation entitled **PREVALENCE OF GROUP B STREPTOCOCCI IN PREGNANT WOMEN AND THEIR NEONATES** was prepared by me under the direct guidance and supervision of Prof. Dr. Seetha Panicker, MD, DGO, DNB. PSG Institute of Medical Sciences and Research, Coimbatore.

The dissertation is submitted to the Dr. MGR Medical University in fulfillment of the University regulations for the award of MD degree in Obstetrics & Gynaecology. This dissertation has not been submitted for the award of any other Degree or Diploma.

Dr. Alphy S. Puthiyidom

ACKNOWLEDGEMENTS

No academic work is single handedly accomplished. This work is no exception. Words fail me in expressing my heart felt and humble gratitude to my guide **Prof. Dr. Seetha Panicker** ,HOD, Department of Obstetrics & Gynaecology, PSG Institute of Medical Sciences & Research for guidance and encouragement all along in completing my study. I acknowledge the kind and willing cooperation extended to me by **Prof. Dr. T.V. Chitra** and **Prof. Dr. Reena Abraham**, Department of Obstetrics & Gynaecology, PSG Institute of Medical Sciences & Research.

I am so grateful to the Principal **Dr. S.Ramalingam** and Medical Directors **Dr. Vimalkumar Govindan** and **Dr. Radhakrishnan** for permitting me to carry out this study.

I am so grateful to **Prof. Appalaraju**, Head of the Dept. of Microbiology and **Dr.Saira**, Asistant Professor, Department of Community Medicine.

I devote this work to my husband **Dr David Jacob** for his valuable help to complete this study.

I am indebted to all teaching staff and colleagues of my department for their valuable suggestions and auxiliary attitude. I am extremely thankful to all the patients who were the most important part of my study. I pray for their longevity.

CONTENTS

INTRODUCTION

Streptococci have an important position in clinical medicine as human pathogens. Among them Group A *Streptococci* have had a special place as a causative agent of important clinical disease recently. Other members of this family are now coming to light as human pathogens and Group B *Streptococci* (GBS) have emerged as important pathogens within the last few decades.

The GBS are known to cause a wide variety of infections in adults, but clinical interest in these bacteria mainly relates to their ability to cause serious neonatal illnesses especially meningitis and sepsis. In developed countries these organisms are the leading cause of neonatal sepsis and meningitis with a case fatality rate of 40 -80 %. The reason for this is not clear.

Streptococcus agalactiae is the only species that carries the Group B antigen. This organism was initially recognized as a cause of puerperal sepsis. Although this disease is now relatively uncommon, *S. agalactiae* has become better known as an important cause of septicaemia, pneumonia and meningitis in newborn children as well as a cause of serious disease in adults.

Group B *Streptococci* are Gram-positive cocci (0.6 to 1.2 micrometre size) that form short chains in clinical specimens and longer chains in culture; features that make them indistinguishable on Gram stain from *Streptococcus pyogenes*. They grow well on nutritionally enriched media and in contrast with

the colonies of *Streptococcus pyogenes,* the colonies of *Streptococcus agalactiae* are buttery with a narrow zone of beta haemolysis. Some GBS strains (1 -2%) are non-haemolytic, although their prevalence may be underestimated because non-haemolytic strains are not commonly screened for the group B antigen.

Strains of *Streptococcus agalactiae* can be characterised on the basis of three serologic markers.

1. The B antigen or Group-specific Cell Wall Polysaccharide antigen.
2. Type-specific Capsular Polysaccharide Antigen (Ia, Ia/c, Ib/c, II, IIc, II to Viii)
3. The Surface Protein or c protein.

Any person can carry GBS, but few become sick from it. The bacterium lives in the lower genital tract or gastro-intestinal system along with many other bacteria that are harmless to most people. The bacterium causes illness primarily in pregnant women and their babies and occasionally in the elderly and in adults with other illnesses such as cancer and diabetes.

If a pregnant women carries the GBS bacterium in her vagina or rectum at the time of labor and delivery, there is a 1 in 100 chance that her baby will become sick from GBS infection. The risk rises to 4% if a pregnant woman carries the bacterium and also has certain risk factors. These risk factors

include-

- Pre-term delivery before 37 weeks gestation
- Prolonged rupture of membranes (longer than 18 hrs without delivering the baby)
- Fever (100.4^0F or higher) during labour

Other risk factors include having a previous pregnancy resulting in a GBS- infected baby or having a urinary tract infection caused by GBS.

Babies who become sick with GBS infection take the bacterium into their bodies by ingesting GBS-containing amniotic or vaginal fluids during labor and delivery.

There are two forms of GBS infection in infants - Early-onset and Late-onset. Babies with Early-onset infection develop symptoms within seven days of birth; most commonly with in the first day of life. Babies with a late-onset infection develop symptoms between seven days and 3 months of age.

About 80% of all GBS infections in newborns are Early-onset infections. Early-onset infections are almost always transmitted from mother to baby around the time of delivery. Late-onset infections can be contracted at delivery or acquired after birth from contact with the mother or other people who are GBS carriers. Babies with an Early-onset infection suffer from one or more conditions like Pneumonia, Sepsis (Blood infection) and less commonly

Meningitis. Babies with Late-onset infections usually have Sepsis or Meningitis.

In spite of treatment with antibiotics, about 5% of babies with GBS die. Preterm babies are more likely to die from the illness than are full term babies. Most babies who survive GBS go on to develop normally. However among these who develop meningitis, up to 5 % suffer lasting nuerologic damage that can include cerebral palsy, sight and hearing loss, mental retardation, learning disabilities and seizures.

According to the 2002 guidelines of the Centers for Disease Control and Prevention (CDC), United States of America, all pregnant women should be screened for GBS at 35-37 weeks of pregnancy. The health care provider is to take a swab from the vagina and rectum and send the sample to a laboratory for a culture to test for the presence of GBS. The results are usually available in 24-48 hours. If a pregnant woman is found to carry GBS, she should be treated with intravenous antibiotics during labour and delivery. A recent CDC study showed that this approach may prevent nearly 90% of Early-onset GBS infections. Taking oral antibiotics prior to labour is not recommended as it is not effective in preventing GBS infection in the newborn.

The vaccination of women of child-bearing age and the provision of protection by placental transfers of specific antibody appears to be an ideal

solution for the prevention of neonatal GBS sepsis. Vaccine trials in pregnant women would also be ethically difficult. As GBS disease is still a relatively rare event, large sample sizes would be needed to show an effect on neonatal sepsis rate.

In addition to potentially reducing neonatal GBS disease, vaccination could also lead to reductions in maternal GBS urinary tract infection and chorio-amnionitis and could potentially prevent preterm birth. Also, if vaccination were a reality, the complications associated with intra-partum antibiotic prophylaxis such as maternal anaphylaxis, antibiotic resistance and changes in neonatal sepsis rates would no longer present the same potential problems.

AIMS OF THE STUDY

1. To find out the prevalence of Group B *Streptococci* colonization in pregnant women at the time of admission and in their neonates.

2. To find out the association of maternal colonization with Group B *Streptococci* in pregnancy and maternal complications like Premature Rupture of Membranes (PROM), Preterm labor, Maternal fever, Puerperal sepsis.

3. To correlate the association of swab positivity of Group B *Streptococci* in neonates and neonatal complications like sepsis, pneumonia and meningitis.

REVIEW OF LITERATURE

Group B *Streptococci* is an important cause of neonatal infection in the Western Hemisphere. The recognition that the maternal colonization with the organism is a key factor in the occurrence of GBS associated neonatal morbidity and mortality was a milestone in the history of perinatal health[1]. A nation-wide change in health practices helped diminish mortality and morbidity associated with the disease. In India, however, the spectrum of GBS disease remains a largely under-recognized problem.

Puerperal sepsis has been described for centuries and ancient Indian texts in 1500 BC have recorded that good hygiene leads to a reduction in perinatal disease[2]. In 1879 Louis Pasteur identified *Streptococcus* as the causative organism for puerperal sepsis [3]. Since the early 1930's when Rebecca Lancefield proposed a grouping system for hemolytic *Streptococci*, Group A *Streptococcus – Streptococcus pyogenes* was widely acknowledged as the major pathogen associated with puerperal sepsis[4]. GBS was initially thought to be a commensal until 1937, when Fry reported 7 cases of GBS-associated puerperal fever with 3 deaths [5].

There is a spectrum of maternal and fetal GBS infections ranging from asymptomatic colonization to sepsis. *Streptococcus agalactiae* has been implicated in adverse pregnancy outcomes, including preterm labor, prematurely ruptured membranes, clinical and sub-clinical chorioamnionitis, and fetal and neonatal infections. The bacterium can also cause bactriuria, pyelonephritis, and postpartum metritis. Barbosa-Cesnik *et al.* 2003 have reported postpartum maternal osteomyelitis [6] and Berkowitz and McCaffrey, 1990 have reported mastitis by GBS [7].

A higher prevalence has been found in younger women. . GBS is not considered a sexually transmitted infection and treatment of partners does not prevent re-colonisation of treated women. Throughout pregnancy colonization can be transient, intermittent or chronic [8].

GBS SEROTYPES AND PATHOGENESIS

There are 9 antigenically distinct serotypes based on their capsular polysaccharide structure (types Ia, Ib, II-VII) identified to date. In the United States and Western Europe, types Ia, II and III accounted for 85 per cent of the isolates from infants [9,10]. Recent studies in the US have demonstrated that Serotypes Ia, III and V (in descending frequency) accounted for 78 – 87 per cent of early-onset (less than seven days after birth) invasive disease in

newborn infants and parturient women [11,12]. Late-onset GBS disease in infants 7 -90 days of age is dominated by serotype III, followed by serotypes Ia and V [12].

Studies from India show a variable distribution of serotypes. But the most common isolates belong to types III, II and Ib [13,14, 15].

The most important risk factor for early-onset GBS infection in the neonate in the presence of the organism in the maternal genitourinary tract at the time of delivery. Ascending bacteria from the maternal genital tract reach the amniotic fluid, usually after rupture of the amniotic membranes [1, 16]. Alternatively the newborn can come into contact with GBS during passage through the birth canal. When the foetus aspirates contaminated amniotic fluid, GBS reach the lower respiratory tract and damage pulmonary epithelial cells, resulting in pneumonia and respiratory distress usually within the first few hours after birth. Severe GBS sepsis occurs with intravascular invasion of bacteria and failure of the host to eliminate the pathogen [17, 18]. Ascending infection can also occur through intact chorioamniotic membranes, with subsequent events occurring in utero, resulting in still births or death within hours after birth [16]. The pathogenesis in late-onset disease is less clear. Horizontal transmission plays a major role, such as by close contact with the mother, breast-feeding and nosocomial

transmission.

The polysaccharide capsule is the most important virulence factor [18]. However, the role of surface localized GBS proteins in pathogenesis and protection is under intensive investigation [18]. The presence of maternal serum antibodies to specific capsular polysaccharides of GBS serotypes appears to be protective against acquisition of neonatal GBS disease as colonized pregnant women with high levels of serum antibodies were less likely to have neonates with invasive GBS infection [19, 20]. Moreover, infected infants had low levels of specific antibody to the infective serotype [21].

PREVALENCE OF GROUP B *STREPTOCOCCI*

During the 1970's and 1980's GBS emerged as a major pathogen in the United States and Western Europe with reported mortality rate of 15% - 50 % [22, 23]. In US 10-30% pregnant women are asymptomatic carriers of GBS in the genital and gastrointestinal tract at the time of delivery. The prevalence of GBS colonization in pregnancy is variable [24] .

Boyer KM, Gadzala et al (1982) reported that women who had positive GBS cultures between 26 and 28 weeks gestation only 65% remained colonized at term while 8% of those with negative prenatal cultures were positive for GBS at term [25]. Treatment of these colonized

mothers succeeded in temporarily eradicating the organism, but most of the women were recolonised within 6 weeks. At birth 50 – 65% of infants who are born to colonized mothers have positive GBS cultures from mucus membranes and skin[26]. Approximately 98% of colonized newborn remain healthy. But 1–2% had invasive GBS infection. The overall incidence of neonatal GBS infection was approximately 2/1000 live births in United States prior to the introduction of intrapartum prophylaxis [23]

Scrag and associates (2002 – 2003) reported a colonization rate of 20 – 30% in a nation wide cohort sampled at a mean of 35 weeks [27] .

El-Kersh TA, Al-Nuaim LA et al (2002) studied the carrier state of GBS in Saudi females during 3rd trimester of pregnancy . This study included 217 pregnant women and documented a GBS colonization rate of 27.6%. Additionally, 50% of GBS colonized mothers vertically transmitted the organism to their newborns [28].

Orrett FA. *et al.* analyzed 201 third trimester pregnant women of Trinidad and Tobago, West Indies. The prevalence of vaginal and rectal GBS colonization was 32.9%. GBS were isolated more frequently from women aged more than 24 years (36.6%) than those younger than 24 years (26.9%). Colonization rates were significantly greater among multigravid women than primigravid women.

Dillon HC Jr, Gray E, *et al* (1982) did a longitudinal prospective study of carriage of GBS during pregnancy in 2,500 women over a three-year period. Carriage was documented in 18% of the women by anorectal culture, in 4 % by vaginal culture, and in 13 % by simultaneously obtained anorectal and vaginal cultures (Overall carriage rate, 35%). The intestinal tract appeared to be a primary reservoir for colonization in pregnant women [29].

Terry RR Kelly FW. *et al*, (1999) did a study on 608 pregnant women between 1995 and 1997 to consider a number of possible risk factors for GBS. 14.0% of the study subjects were found to be colonized with GBS. White non-Hispanic women had a GBS colonization prevalence of 13.6%; for all others, prevalence was 18.7%. No statistically significant differences were found in regard to age, weight, number of prenatal visits income level, marital status, history of drug use, or parity. This study identified smoking as a possible risk factor for GBS infection with the GBS colonization rate for smokers was 33.1% versus 16.4% for nonsmokers. The authors concluded that routine screening for GBS infection during pregnancy may be beneficial because no strong risk factors for colonization exist [30].

Eren A, Kucukercan M, *et al.* (2005) studied 500 Turkish pregnant women and their newborn infants by collecting vaginal and rectal swabs from mothers, and umbilical and throat swabs from their infants. Maternal

and infant colonization rates were found to be 9.2 % and 1.6 %, respectively. Vertical transmission rate was 15.2 %. Although invasive serotypes were predominant, the rarity of GBS disease in their study was thought to be due to low rates of maternal carriage or to their possessing protective levels of GBS- specific IgG antibody in their sera [31].

Gerard P, Verghote–D'Hulst M, *et al* did an epidemiological study and controlled trial of prophylactic treatment of the newborn. Colonization with GBS of the genital tract was studied in 1115 women during the last trimester of pregnancy. 6.82 % of women were found to harbour this bacterium. It was also more frequent in primigravidae. Rupture of the amniotic membranes for more than 24 hours was more often associated with GBS carriage by the mother. 42.6% of the infants born to GBS positive mothers were colonized at birth. The study also indicated that immediate therapy with penicillin of infants of GBS positive mothers has no definite advantage upon delayed treatment [32].

Schrag SJ, Zell ER *et al* in their multistate retrospective cohort study they compared the effectiveness of the screening and risk-based approaches in preventing early-onset GBS disease in a sample of 629, 912 live births in 1998 and 1999. Antenatal screening was documented for 52 percent of the mothers. The risk of early-onset disease was significantly lower among the

infants of screened women than among those in the risk-based group. So routine screening for GBS prevents more cases of early-onset disease than the risk-based approach [33, 34, 35, 36, 37].

Al-Sweih N, Maiyegun S *et al* (2004) Kuwait University, studied the prevalence of GBS in Kuwait population. Anal, vaginal and combined anal and vaginal specimens were obtained from pregnant women at 35-37 weeks of gestation. The combined vaginal and anal specimens were positive for GBS in 6.4 % of women. [38].

PREVALENCE OF GROUP B *STREPTOCOCCI*

Year of Study	**Population studied**	**Sample Size**	**Maternal Prevalence**
El-Kersh TA, Al-Nuaim LA et al (2002)	Saudi Arabia	217	27.6 %
Orrett FA. *et al.* (2004)	West Indies	201	32.9 %
Dillon HC Jr, Gray E, *et al* (1982)	USA	2500	35 %
Terry RR, Kelly FW. *et al*, (1999)	USA	608	14 %
Eren A, Kucukercan M, *et al*. (2005)	Turkey	500	9.20%
Gerard P, Verghote–D'Hulst M, *et a*l (1979)	Belgium	1115	6.82 %
Al-Sweih N, Maiyegun S	Kuwait	110	6.4 %

et al *(*2004)			

STUDIES IN INDIA

Epidemiological studies in India have shown lower colonization and infection rates in general [13, 39, 40, 41].The reason is not known or the problem has not been adequately studied. There are only a few reports available however on closer analysis.

Stoll BJ, Schuchat A. (1998) reported that with the use of adequate culture techniques and microbiological media some of the GBS colonization rates reported from India and other developing countries are similar to those reported in United States [39].

Kulkarni AA, Pawar SG, *et al*, Maharashtra studied the prevalence of GBS colonization and its risk factors in 317 pregnant women at the time of labour and in their neonates in 1998-1999. The GBS colonization rate in pregnant women at labour and in neonates was 2.52% and 1.26% respectively with a frequency of transmission of 50%. Only one risk factor has been seen in two GBS colonized mothers (i.e. in one case premature delivery and in the other case premature rupture of membranes) to be

associated with GBS transmission . All isolates were sensitive to Ampicillin, Erythromycin and Penicillin followed by Chloramphenicol 66.6% (12/18). All isolates were resistant to Gentamicin, followed by Tetracycline (17/18) 94.4%, and Kanamycin (16/18) 88.8% [42].

Mhaskarrita, Sathyan Sharad *et al* (2005) St. John's Medical College Hospital Bangalore had done a Selective risk factor based screening of pregnant women for GBS colonization. A retrospective analysis was done for the occurrence of GBS colonization among 741 pregnant women who were at risk i.e. they had at least one of the following risk factors namely- prolonged rupture of membranes (>18 hours), preterm labor (<37 weeks), intrapartum fever, vaginal discharge, and previous baby with GBS infection, Vaginal swab and urine cultures indicated GBS. The occurrence of neonatal GBS infection was also studied.

The occurrence of GBS colonization was 1.62% and neonatal GBS infection was 0.53 per 1000 live births. [43,44,45, 46] .

Goyal R, Singh NP *et al*, GBS was examined in 304 pregnant women in Delhi, [2004]. Vaginal specimens were collected and examined for GBS. GBS was isolated from only 4 women (1.3%). It is suggested that GBS infection is not a problem in this population, and mass screening for GBS

during pregnancy is not needed [47].

Dalal BS, Lahiri A *et al* (1998) did a study on 507 pregnant Indian women; 12 per cent were reported to have GBS isolated from the throat and vagina, and 10 per cent had positive vaginal cultures alone [48].

Chaudhary U, Sabherwal U *et al* have reported colonization rates of 5 to 6 per cent, but no selective broth media were used in these cases [13].

PREVALENCE OF GROUP B STREPTOCOCCI IN INDIA

Year of Study	**Population studied**	**Sample Size**	**Maternal Prevalence**
Kulkarni AA, Pawar SG, *et al* (2001)	Govt.Medical College Maharashtra Miraj	317	2.52 %
Mhaskarrita et al (2005)	St. John's Medical College, Bangalore	741	1.62 %
Goyal R, Singh NP (2004)	Guru Tegh Bahadur Hospital, Delhi	304	1.3 %

Jamie WE, Edwards RK *et al* (2004) performed a prospective cohort study to determine whether the rates of recovery of Group B *Streptococci* from vaginal and perianal cultures and combined vaginal and rectal cultures are equivalent. 36% had a positive culture from at least 1 site. A significant

finding was that the detection rate of Group B *Streptococci* from combined vaginal-perianal specimens is not significantly different from the detection rate from vaginal-rectal specimens. The conclusion was that pregnant women need not be subjected to the discomfort of collection of a rectal specimen [49,50,51].

GROUP B *STREPTOCOCCAL* INFECTION IN NEONATES

It is estimated that about half of all babies born to women carrying GBS will themselves be colonized with GBS. However, the vast majority of these infants will not develop symptomatic GBS infection. A nation-wide study involving active surveillance of infants younger than 90 days showed 377 cases of confirmed early-onset GBS neonatal sepsis out of a total population of 794, 037 live births. It was calculated that the risk of early-onset neonatal sepsis developing as a result of being colonized is about 5 per 1000 [52.]

Recent evidence has suggested that the incidence of culture-proven GBS neonatal sepsis is likely to underestimate the true incidence of this infection in neonates. Data collected prospectively in one centre in the UK for over a year for 413 neonates who underwent a septic screen in the first 72h after birth have suggested that the true incidence of early-onset neonatal

GBS sepsis may be as high as 3.6 per 1000 live births [53]. This was based on the presence of GBS colonization and symptomatic neonatal sepsis without isolation of any organism from a usually sterile site such as blood or CSF.

In the US, the incidence of culture-confirmed, early-onset GBS neonatal sepsis was 2-3 cases per 1000 live births in the early 1980 [54]. Following the introduction of a national screening and treatment programme advocated by the centers for disease control and prevention, the incidence of confirmed GBS neonatal sepsis has fallen to 0.32 per 1000 live births which is the lowest ever recorded (Fig.1) [55, 56]. In contrast, the rate of late-onset GBS sepsis has remained fairly constant at 3.35 per 1000 live births over this period, leading to the suggestion that nosocomial infection plays a large part in the etiology of late-onset disease [56].

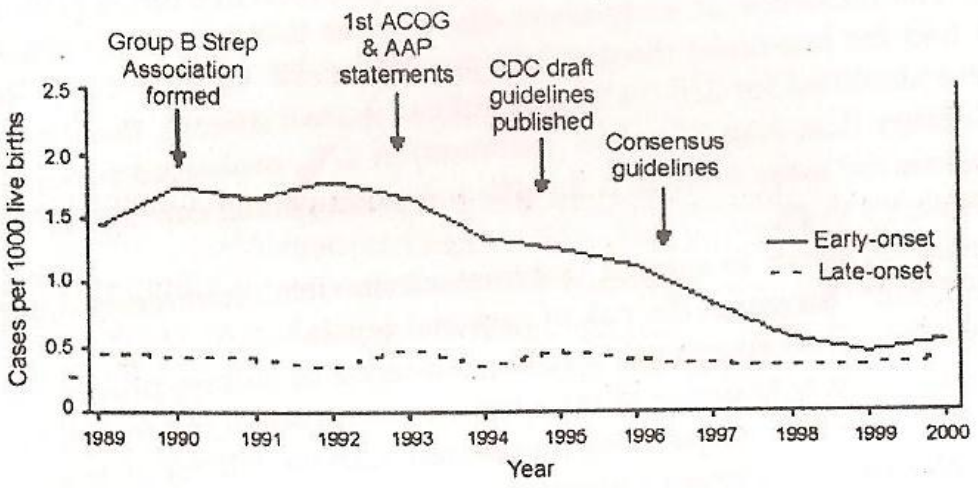


Fig. 1 Incidence of early- and late-onset invasive GBS disease – selected Active Bacterial Core surveillance areas, 1989–2000, and activities for prevention of GBS disease. ACOG, American College of Obstetricians and Gynecologists; AAP, American Academy of Pediatrics. Adapted from CDC (Early onset group B streptococcal disease, United States, 1998–1999. *MMWR* 2000; 49: 793–796) and Schrag *et al*.

NEONATAL MORBIDITY

Two distinct clinical syndromes are recognized, early-and late onset disease. Early onset GBS disease occurs within the first 7 days of life, although most cases are evident in the first 24 h after birth. As can be demonstrated by serotyping GBS isolates from colonized mothers and infants, transmission of early-onset disease is vertical [25]. Infection may be acquired by the intraammiotic route, or directly during passage through the birth canal. The initial presentation is respiratory distress in more than 80 percent of neonates [57]. Pneumonia and septicaemia are the most common manifestations and 5 to 10 percent neonates will also have meningitis. The

incidence of early-onset disease is about 10 times higher in premature than in term neonates. Late-onset disease develops in infants after 7 days and up to 3 months of age, the median age of onset being 1 month. Transmission can be either horizontal (from other infected infants or health care workers) or vertical (from the mother due to close proximity). These infants almost always have an unremarkable early neonatal history, and later present with meningitis or sepsis Osteoarticular infections and cellutitis can also occur [58]. The case fatality rate in the US has dramatically decreased over the last 3 decades, from up to 50 per cent in the 1970s to 6 per cent in the early 1990s [59].

Currently most centers and countries have experienced a decline in early –onset neonatal sepsis to rates of less than 1 to 2 per 1000 live births. Most reports, however, indicate no change or variable increases in rates of non-GBS organisms, such as *E. coli* and other Enterobacteriaceae [60, 61, 62, 63.]

There is evidence that the major association with current intrapartum antimicrobial prophylaxis has been an increase in non-GBS early–onset sepsis in preterm, low-birth weight neonates, and especially very-low-birth weight neonates [63, 64]. Stoll and associates (2002) observed a marked reduction in Group B streptococcal sepsis in these preterm neonates; this was offset by an increase in E coli sepsis.

Late-onset neonatal GBS sepsis is less well understood. Cited rates vary from 0.5 to 2 cases per 1000 live births and account for about 50 percent of GBS disease in newborns [65,66]. The incidence of late-onset disease has remained stable despite widespread use of intrapartum antimicrobials, suggesting that GBS screening and chemoprophylaxis intervention may not affect late-onset disease. Lin and co-workers (2003) reported that preterm birth before 34 weeks was the major risk factor [66]. Stoll BJ, Hansen N (2002) found that the late-onset sepsis was identified in a fourth of very-low-birth weight newborns; there was a preponderance of gram-positive organisms, mainly coagulase-negative staphylococci.

The most recent report from the CDC 2002 reports mortality rate of 0.7 per 100,000 population [67]. Case series from India also report high mortality. In a 1975 study where 8 cases of neonatal GBS infection were followed over a period of 18 months, 6 infants died within the first week of diagnosis [68]. A more recent study published in 1999 reported 10 infants with GBS infection, of which 1 died on the second day of life. However, there is insufficient data presented to calculate the actual mortality rate [69].

Kuruvilla KA, Thomas N *et al* (1999) The incidence of GBS bacteraemia was 0.17 per 1000 live births. Lethargy, respiratory distress and poor perfusion were the presenting features in eight symptomatic babies.

Two babies had meningitis, three required ventilatory support and one died. There were no cases of late onset disease. The low incidence could be due to the low rate of colonisation and high prevalence of protective antibody in the mothers.

The estimated incidence of neonatal GBS infection in India can be calculated from Indian epidemiological data reporting maternal and infant GBS colonization rates as 10 and 5 per cent respectively. Since about 2 per cent of colonized neonates develop true infection, the attack rate of neonatal GBS infection in India may be calculated as approximately 1 per 1000 live births. Bearing in mind the above estimated attack rate and current Indian demographic data (midyear population count in the year 2001 was approximately 1027 million, and birth rate was 26 births per 1000 population per year), the projected total number of GBS infection in newborn infants in India may be a s high as 26,700 cases per year [70].

Strakova ,Motlova *et al* (2003) isolated GBS from 239 full-term and 46 preterm newborns. They reported the incidence of Early onset disease due to GBS in Czech Republic is 0.7-1.0 per 1000 live births [71].

NEONATAL PREVALENCE

Year of Study	**Population studied**	**Sample Size**	**Maternal Prevalence**

Kuruvilla et al 1999	CMC Vellore	60119	0.17/1000 live birth
Kulkarni & pawar etal 2001	Govt.Medical College. Miraj	317	1.26 %
Mahskarrita etal 2005	St.John's Medical College Bangalore	741	0.53 / 1000 live birth
National Surveillance Study	UK	568	0.72 / 1000 live birth
Strakova & Motlova etal 2003	Czech Republic	285	0.96 / 1000 live birth

Klebanoff MA, Regan JA, and associates (1995) did a double-blind study to determine whether Erythromycin treatment of pregnant women colonized with GBS would reduce the occurrence of low birth weight. Erythromycin or placebo was given to pregnant women beginning during the third trimester and before 30 weeks and continuing for 10 weeks or until 35 weeks 6 days of pregnancy. Study concluded that treating pregnant women colonized with GBS with Erythromycin was not effective at prolonging gestation or reducing low birth weight [72].

Kurien Anil Kuruvilla, Swati Pillai etal studied on babies born in the Christian Medical College Hospital (CMCH), Vellore,(1995-1996) .Infants

with clinical signs of sepsis or those who were born to mothers with potential risk factors for infection were screened for sepsis. Risk factors in the mother for EOS included prolonged rupture of membranes (PROM >24 hours), maternal pyrexia, untreated urinary tract infection (UTI), chorioamnionitis, or multiple vaginal examinations.The incidence of neonatal bacterial sepsis is 9.8 per 1000 livebirths. *E. Coli* and *Klebsiella* were the most common organisms causing EOS and LOS. *Enterococcus fecalis* was also a major pathogen, both in EOS and LOS [73].

OTHER ORGANISMS ASSOCIATED WITH GENITAL TRACT INFECTION

Any infection can lead to preterm labour and premature rupture of membranes .Recently lower genital tract infections has been associated with preyerm labour .one of the important issue being highlighted is bacterial vaginosis .Bacterial vaginosis describes a polymicrobial alteration of vaginal flora resulting from overgrouth of the anaerobic bacteria and gardenella vaginalis than lactobacillus .The major bacterial vaginosis associated organisms are *Gardenella vaginalis*, anaerobic gram negative rods, *Bacteroides*, *Peptostreptococcus*, *Mycoplasma hominis* and *Ureaplasma urealyticum*.

PREVENTION STRATEGIES

Lacking randomized trials, consensus opinions and guidelines on prevention strategies have been promulgated by the American College of Obstetricians and Gynecologists (2002) and the Centers for Disease Control and Prevention (2002). These guidelines advocate a culture –based screening approach to identify women who should receive intrapartum prophylaxis. This recommendation was derived from a multistate, retrospective cohort study of live births in 1998 and 1999 from the Active Bacterial Surveillance/Emerging Infections program network, which suggested that the culture-based approach was superior to a risk-based approach.

With the culture-based approach, women are screened for GBS colonization at 35 to 37 weeks, and intrapartum antimicrobials are given to rectovaginal carriers. Previous siblings with GBS invasive disease and prior identification of GBS bacteruria are also considered indications for prophylaxis. A risk – based approach is recommended for women with un-known GBS culture results at the time of labor.

The choice of antimicrobials may be important in terms of allergic reaction; selection of resistant GBS strains; and emergence of other pathogens, including antimicrobial-resistant strains, as agents of neonatal

sepsis. The centers for disease control and prevention recommendations specify penicillin as a first-line agent [66]. Ampicillin is an acceptable alternative [73]. For women with penicillin allergy, if the risk of anaphylaxis is low, cefazolin is recommended [74]. If the risk of anaphylaxis is high, selection of a prophylactic agent is dependent on GBS susceptibility testing. Patients with isolates susceptible to clindamycin or erythromycin may be given either drug. Antimicrobial resistant strains require vancomycin prophylaxis. This treatment scheme is dependent on laboratory capability to perform susceptibility testing.

Importantly, there have been no randomized controlled trials comparing antenatal screening. In addition, there have been no randomized trials comparing the different screening strategies and whether prenatal GBS screening has a significant impact on overall neonatal sepsis. For these reasons, clinicians in other countries state that there is in sufficient evidence to recommended screening for GBS carriage [75,76,77].

Alternative prevention strategies have been described with limited evidence to recommend them. These include intramuscular benzathine penicillin G and chlorhexidine vaginal lavage [78]. In another study, Haberland colleagues (2002) reported that intrapartum rapid PCR screening for GBS may be superior to current strategies; however, this must be proven effective

in clinical trails [79]. In GBS- Positive women, appropriate vaginal examinations or indicated intrauterine fetal monitoring should not be avoided as their avoidance could actually prolong labor and thus increase the risk of infection[80].

Implementation of several protocols is associated with diminished GBS. There are, however, still major concerns about antimicrobial resistance, particularly among very-low birth weight neonates. Ongoing surveillance is necessary to monitor protocol efficacy for prevention of early-onset GBS sepsis as well as for any effects on maternal morbidity, overall neonatal sepsis, and resistant infections.

VACCINATION

Some protection against serious neonatal infection is conferred by maternal antibodies. Indeed, Lin and colleagues (2001) have confirmed that the susceptibility to invasive GBS disease correlates with deficiency in maternal type-specific antibody levels [81]. Baker and co-workers reported that maternal immunization to type III antigen produces antibody in about 60 percent of women Monovalent tetanus toxoid conjugate vaccines are immunogenic for common GBS disease –associated serotypes [82,83,84]. Paoletti and Modoff (2002) have reviewed the progress toward development of a multivalent vaccine [85].

MATERIALS AND METHODS

Women admitted to PSG Hospitals with labour pain, preterm labour, premature rupture of membranes were included in the study irrespective of gestational age, parity, or socioeconomic status. Two hundred women and their neonates were included in the study from February 2006 to February 2007.

METHODS

Sample Collection

At the time of admission to the labour Room, two swabs were collected from all pregnant women who were admitted with labour pain, preterm labour, or premature rupture of membranes. Four swabs were collected from all neonates soon after birth.

Maternal Sample Collection

After obtaining the informed written consent, two swabs were collected from each mother-one from the vagina and the other from the rectum.

Vaginal Sample Collection

One swab was collected from the lower vagina taken prior to first

pelvic examination. No antiseptic preparation of the perineum or vulva was carried out before swabbing. Speculum was not used for culture collection.

Rectal Swab Collection

A different swab was inserted through the anal sphincter and a sample was taken.

Both the vaginal swab and the rectal swab were placed in selective broth medium and transported to the laboratory.

Samples from Neonates

A total of four samples were collected from each newborn infant immediately after birth. Swabs were taken from the external ear, nose, throat and umbilical region. These swabs were transported to laboratory after placing the swabs in selecting broth medium. (Todd-Hewitt broth)

Selective Broth Medium

The Selective Broth Medium we used in our study was Todd-Hewitt broth which was supplemented with Gentamycin-8 micrograms/ml and Nalidixic Acid-15 micrograms/ml.

Procedure for Processing the Clinical Specimen for culture of GBS

1. The swab is inoculated into the Todd-Hewitt broth supplemented with Gentamycin-8 micrograms/ml and Nalidixic Acid-15 micrograms/ml.

2. Inoculated Todd-Hewitt broth was incubated for 24 hours at $37^{O}C$ in ambient air
3. The samples were then transferred to 5% Sheep's Blood Agar and incubated for a further 24 hours at $37^{O}C$.
4. Plates were then examined for growth of Group B Streptococci. If no growth was found, the plates were incubated for an additional day and re-examined for growth of the organism. If no growth was found on the second examination, the plates were declared as negative.

Demonstration of Group B *Streptococci*.

GBS – appearance in 5% sheep blood agar

The colony is usually gray, soft, shiny, convex, moist, regular and about 1 mm in diameter and surrounded by a small hazy zone of beta haemolysis.

Gram stain

Gram stain of pinpoint colonies is done to demonstrate the presence of Gram positive cocci arranged in short chains.

Confirmation of GBS

GBS Growth was confirmed by CAMP Test.

Antibiotic sensitivity

The isolates were tested for antibiotic sensitivity by disc diffusion method of Kirby-Bauer. Antibiotics tested were Ampicillin, Erythromycin. Tetracycline, Chloramphenicol and Vancomycin.

Post-Natal Follow-up

All mothers in the study were followed up during the period of admission, and by outpatient clinic visits after discharge, for a period of 45 days for fever, urinary tract infection, and vaginal discharge.

Neonates were followed up during the period of admission, and by outpatient clinic visits after discharge, for a period of 90 days after delivery for late onset infections such as meningitis, sepsis and pneumonia.

RESULTS AND ANALYSIS

In our study samples were taken from two hundred women and their neonates who were admitted to the labour room in PSG Hospital from Feb 2006 to Feb 2007. Maternal swabs were taken at the time of admission to the labour room, and swabs from the neonates were taken after delivery. Both samples were cultured separately. All the samples were from singleton pregnancies.

Caesarean deliveries and those deliveries resulting in intra-uterine deaths were excluded from the study. Mothers and their babies were followed up for a total of ninety days after delivery for maternal and fetal complications of Group B *Streptococcus* infection.

MATERNAL AGE OF STUDY GROUP

The age of the mothers included in the study were analyzed.

Table 1 – SHOWING MATERNAL AGE OF STUDY POPULATION

Age of Patients	No. of cases	Study group in percent
< 19 yrs	12	6 %
20 – 29 yrs	163	81.5 %
> 30 yrs	25	12.5 %

In our study, 81.5% of the patients were in the age group of 20 to 29

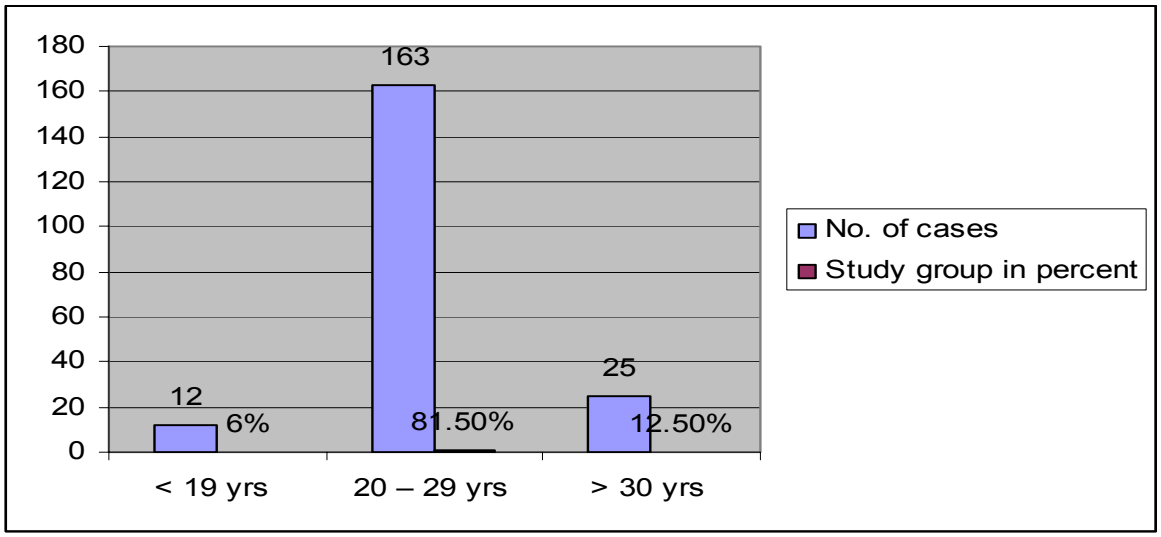


FIG 1 – SHOWING MATERNAL AGE OF STUDY POPULATION

PARITY STATUS

It was found that in our study 52 percent of women were primiparous and 48 percent were multiparous

Table 2 -TABLE SHOWING PARITY STATUS OF PATIENTS

PARITY	NUMBER OF CASES	STUDY GROUP IN PERCENTAGE
PRIMIPAROUS	104	52 %
MULTIPAROUS	96	48 %

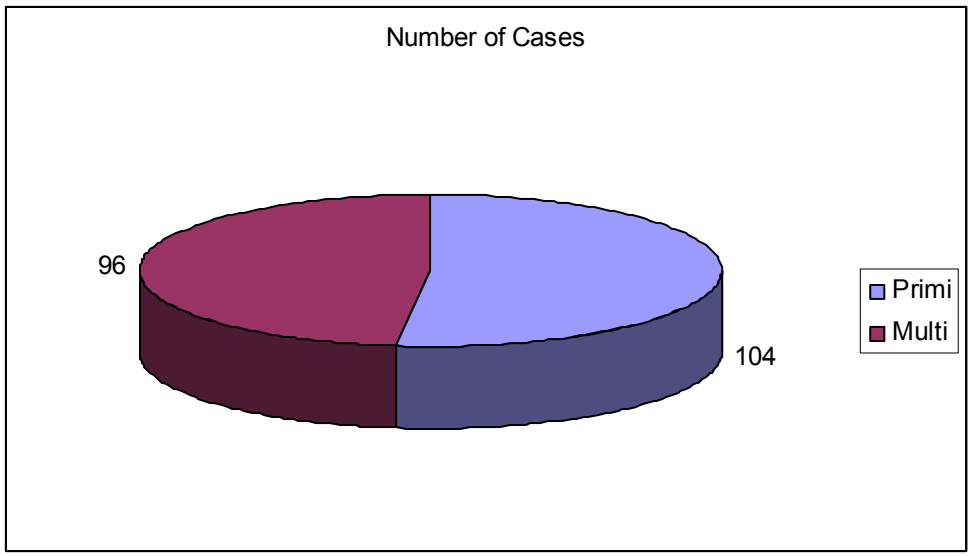


FIGURE 2 – PIE CHART SHOWING PARITY STATUS OF STUDY PATIENTS

SOCIOECONOMIC STATUS

An analysis was done of the socio-economic status of the women enrolled in the study. The classification was based on the occupation of the husband.

Table 3 – SHOWING SOCIO-ECONOMIC STATUS OF STUDY POPULATION

Socioeconomic status	Percentage
Higher Professionals and other professionals – Including those in managerial positions	1 %
Skilled Workers – Non manual	11 %
Skilled Workers – Manual	14 %
Semi –skilled workers	28 %
Unskilled Workers	46 %

It was found that the largest proportion of patients in the study belonged to the lower socio-economic group.

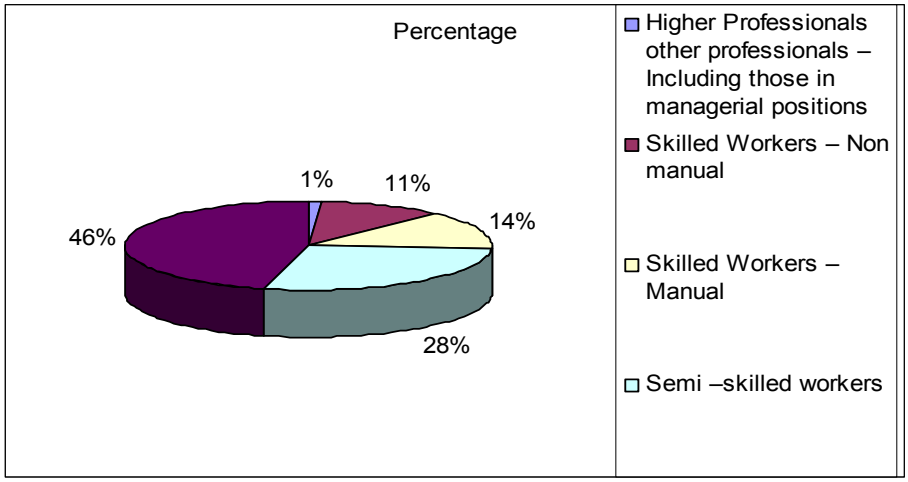


FIGURE 3 – SHOWING SOCIO-ECONOMIC STATUS OF STUDY POPULATION

PREVALENCE OF GROUP B STREPTOCOCCI IN STUDY POPULATION

Maternal Swabs positive for Group B Streptococci

Maternal Swabs	Positive for Group B Streptococci	Negative for Group B Streptococci	Total
Number	4	196	200
Percentage	2	98	100

Table 4 – MATERNAL SWAB POSITIVITY FOR GROUP B *Streptococci*

4 cases making up 2.0 % of the study population were found to be positive for group B Streptococcus

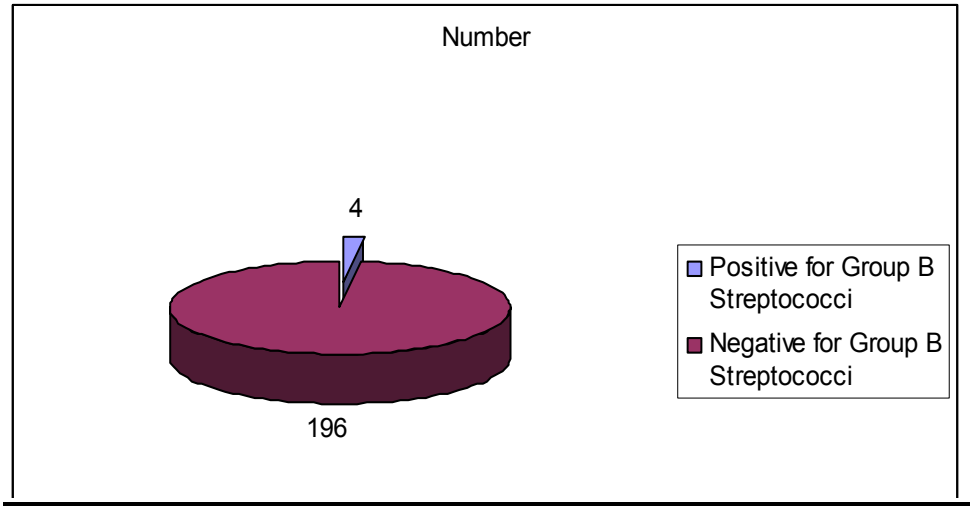


FIGURE 4 - MATERNAL SWAB POSITIVITY FOR GROUP B *Streptococci*

GBS – appearance in 5% sheep blood agar

The colony is usually gray, soft, shiny, convex, moist, regular and about 1 mm in diameter and surrounded by a small hazy zone of beta haemolysis.

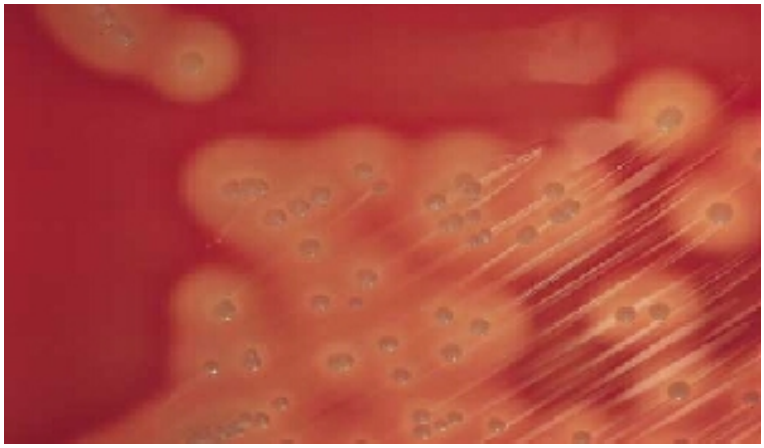


FIG -GBS – APPEARANCE IN 5% SHEEP BLOOD AGAR

GRAM STAIN

Once the colonies were found, Gram stain of pinpoint colonies is done to demonstrate the presence of gram positive cocci arranged in short chains

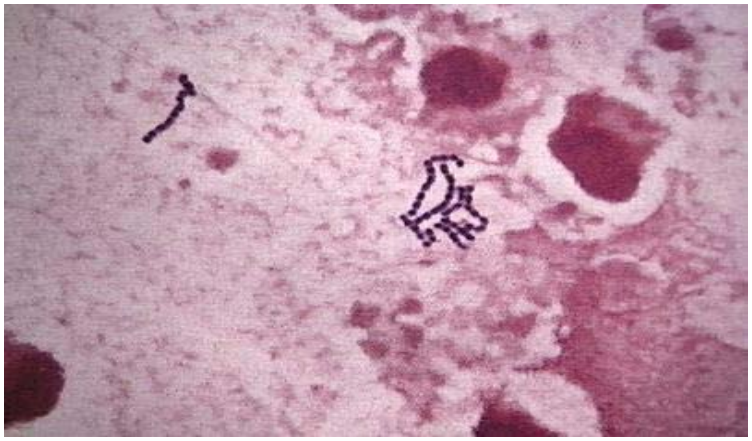


FIG – GRAM STAIN OF GROUP B *STREPTOCOCCI*

Confirmation of GBS

GBS Growth was confirmed by CAMP Test.

CAMP TEST

CAMP (Christie, Atkins and Munch-Peterson) test is used to identify Group B *streptococci*. CAMP substance is a peptide produced by group B *Streptococci* which acts synergistically with the β – hemolysis produced by some strains of *Staphylococcus aureus* enhancing the effect of haemolysis.

The test is performed by inoculating *S.aureus* strain as a straight line on the surface of the sheep blood agar. Known positive control (Group B *Streptococci*), known negative control (group A *Streptococci*) and test strains are also inoculated as a straight lines parallel to *S.aureus* streak line leaving 1 cm space. The plate is incubated aerobically at 37^{O}C overnight in an environment of 5-10% CO_2. Positive test is indicated by an arrow head zone of hemolysis near the *S.aureus* growth. Negative test is indicated by the absence of arrow head zone of hemolysis.

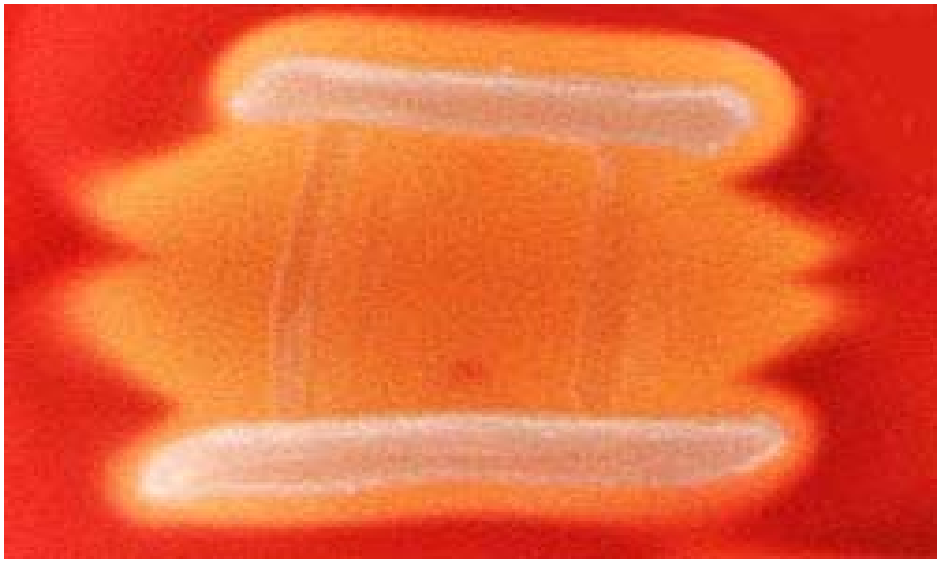


FIG – CAMP TEST

Antibiotic sensitivity

After the growth has been confirmed, the isolates were tested for antibiotic sensitivity by disc diffusion method of Kirby- Bauer

In this method antibiotic sensitivity of the bacteria was determined by disc diffusion method. Standard suspensions of bacteria to be tested are inoculated on the surface of Mueller-Hinton agar plates. Filter paper discs containing specific concentration of antimicrobial agents are pressed on to the surface and incubated at $35^{O}C$ overnight. After incubation, the zone of inhibition of growth of bacteria around each disc is measured and the susceptibility is determined.

Each antibiotics produces a specific zone size for each bacteria tested. Depending on the zone size, the bacteria are classified as follows.

Sensitive (S) : Infection treatable with normal dosage of the antibiotic.

Intermediate (I) : Infection may respond to therapy with higher dosage.

Resistant (R) : Unlikely to respond to the antibiotic at the usual dosage.

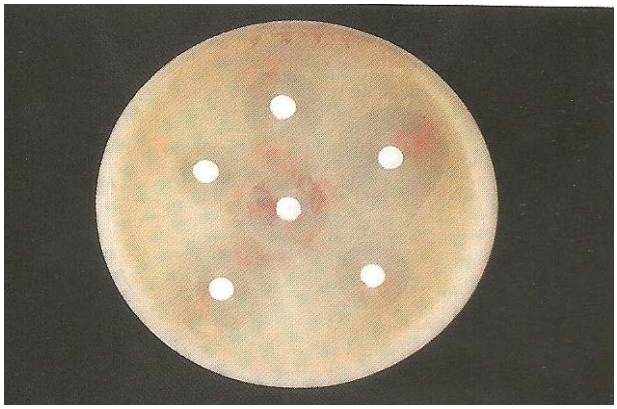


FIG – DISC DIFFUSION TEST

Like the Group A *Streptococci*, isolates of Group B *Streptococci* remain susceptible to penicillin G, the drug of choice for treatment of infections and for perinatal chemoprophylaxis of women who are vaginal carriers of Group B streptococci. Ampicillin, cefotaxime, ceftriaxone, cefezolin, and meropenem are also highly active, with most strains having minimal inhibitory concentrations (MICs) of < 0.06 μg/mL. Some Group B *Streptococci* demonstrate resistance to erythromycin and clindamycin. All group B streptococcal strains tested to data have been susceptible to vancomycin.

Isolates of GBS obtained in our study was sensitive to Vancomycin, Erythromycin, Penicillin, Ciprofloxacin and Tetracycline

and showed resistance to Amikacin and Gentamycin.

BIRTH WEIGHT OF NEONATES

The birth weight of the neonates born during the study was analyzed.

Table 5 - SHOWS THE BIRTH WEIGHT OF THE NEONATES BORN DURING THE STUDY

Birth weight	No of New Born	Percentage
≤2.49 Kg	36	18 %
2.5- 3.49	158	79 %
≤3.5 Kg	6	3 %

79% of the babies born during the study weighed between 2.50 and 3.49 kilograms.

Out of the 36 babies who weighed less than 2.49 kg, 7 babies were from preterm deliveries.

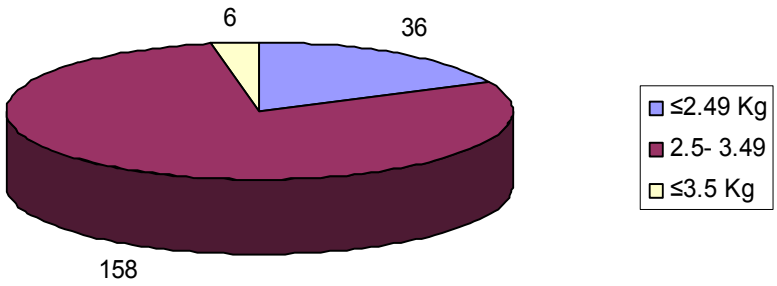


FIGURE 5 – SHOWING THE WEIGHT CATEGORIES OF

NEONATES BORN DURING THE STUDY PERIOD.

GENDER OF THE NEONATES

An analysis was done of the genders of the neonates born to the study population.

Table 6 – SHOWING NUMBER AND PERCENTAGE OF THE GENDER OF THE NEONATES

Sex	No of new Born	Percentage
Male	107	53.5 %
Female	93	46.5 %

It was found that males made up 53.5% of the neonates born during the study.

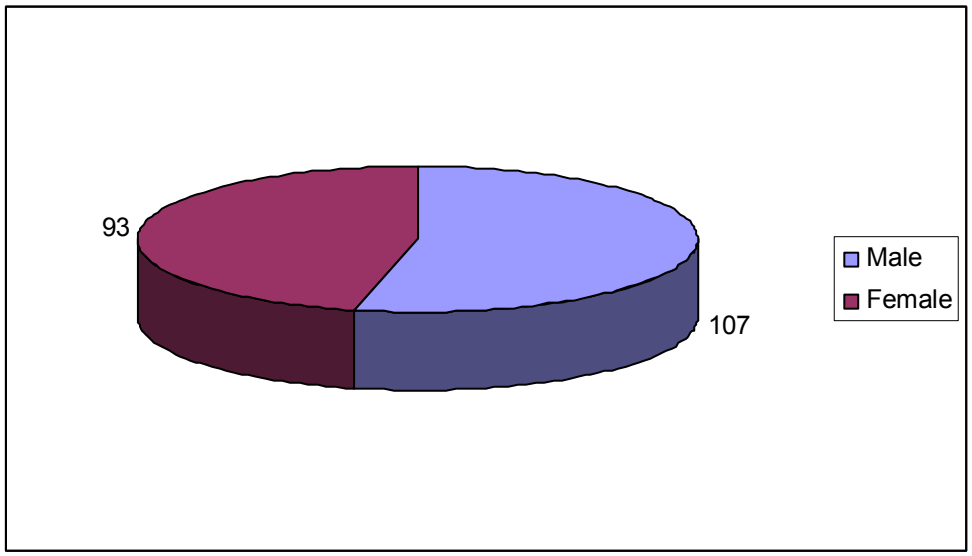


FIGURE 6 – SHOWING PERCENTAGE OF EACH GENDER IN

NEONATES BORN DURING THE STUDY

NEONATAL SWAB POSITIVE FOR GROUP B STREPTOCOCCI

Neonatal Swabs	Positive for Group B Streptococci	Negative for Group B Streptococci	Total
Number	**1**	**199**	**200**
Percentage	**0.5**	**99.5**	**100**

Table 7 -NEONATAL SWAB POSITIVITY FOR GROUP B *Streptococci*

One neonatal swab making up 0.5% of the study population was positive for Group B *Streptococci*.

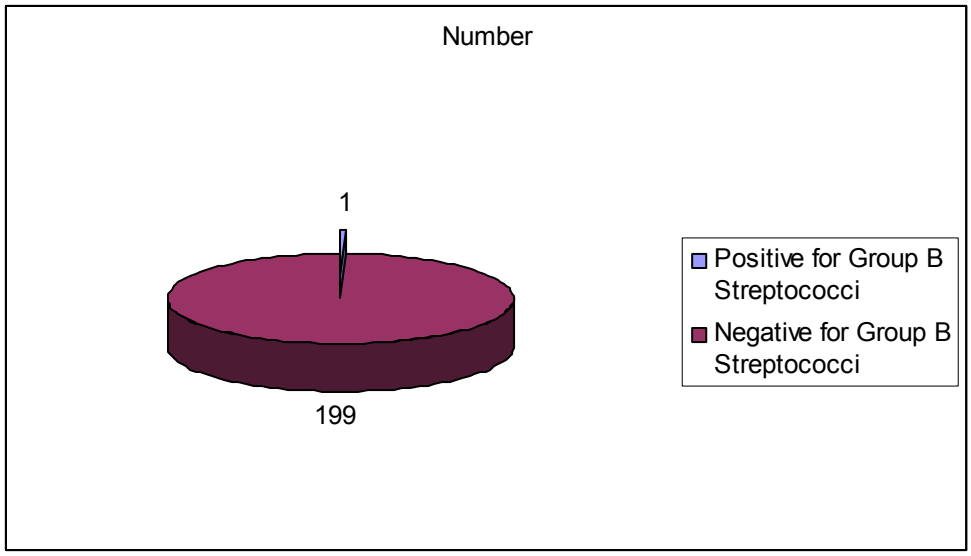


FIGURE 7 - NEONATAL SWAB POSITIVITY FOR GROUP B *Streptococci*

CORRELATION BETWEEN MATERNAL AND NEONATAL SWAB RESULTS.

	Neonatal Swab Positive for Group B *Streptococci*	**Neonatal Swab Negative for Group B *Streptococci***	**Total**
Maternal Swab Positive for Group B *Streptococci*	1	3	4
Maternal Swab Negative for Group B *Streptococci*	0	196	196

Total	1	199	200

Table 8– SHOWING CORRELATION BETWEEN MATERNAL AND NEONATAL SWAB RESULTS

It was found that one neonatal swab in the study was positive for Group B *Streptococci*, and the swab taken from the mother of this child was also positive for the bacterium.

In another 3 cases, the maternal swab was found to be positive for Group B *Streptococcus*, while the swabs from the neonates born to those mothers were negative for the bacterium.

Group B Streptococci Maternal Positivity and Maternal Outcome

Maternal Swab Positivity for Group B *Streptococci* and Maternal Fever

Group B *Streptococci* in Maternal Swab	**Maternal Fever present**	**Maternal Fever Absent**	**Total**
Present	0	4	4

Absent	6	190	196
Total	6	194	200

Table 9 – MATERNAL SWAB POSITIVITY AND MATERNAL FEVER

In the 4 cases that were positive for group B Streptococci in the vaginal swabs, maternal fever was found in none of cases.

It was also found that maternal fever was found in 6 of cases where maternal swabs were negative for group B streptococci.

It therefore appears that other organisms and possibly, other foci of infection, which were not tested for in the present study, are important causes of maternal fever in the peripartum period.

MATERNAL SWAB POSITIVITY FOR GROUP B *STREPTOCOCCI* AND VAGINAL DISCHARGE

Group B *Streptococci* in Maternal Swab	**Vaginal Discharge present**	**Vaginal Discharge Absent**	**Total**

Present	1	3	4
Absent	3	193	196

Table 10 – MATERNAL SWAB POSITIVITY AND VAGINAL DISCHARGE

Vaginal discharge was present in one case where maternal swabs were positive for group B streptococci and in three cases where maternal swabs were negative for Group B Streptococci

MATERNAL SWAB POSITIVITY FOR GROUP B STREPTOCOCCI AND MATERNAL URINARY TRACT INFECTION

Group B Streptococci in Maternal Swab	**Urinary Tract Infection present**	**Urinary Tract Infection Absent**	**Total**
Present	0	4	4

Absent	8	188	196
Total	8	192	200

Table 11 – MATERNAL SWAB POSITIVITY AND MATERNAL URINARY TRACT INFECTION

Maternal Urinary Tract Infection was absent in all cases who were positive for group B Streptococci and in 4 % percent of cases where maternal swabs were negative for Group B Streptococci.

MATERNAL SWAB POSITIVITY FOR GROUP B STREPTOCOCCI AND PRETERM LABOUR

Group B Streptococci in Maternal Swab	Preterm Labour present	Preterm Labour Absent	Total
Present	1 (0.5%)	3 (1.5%)	4(2.0%)

Absent	7 (3.5%)	189 (94.5%)	196 (93.0%)
Total	8 (4.0%)	192 (96.0%)	200 (100.0%)

Table 12– MATERNAL SWAB POSITIVITY AND PRETERM LABOUR

Preterm Labour was present in 25 percent of cases where maternal swabs were positive for Group B Streptococci and in 3.5 % percent of cases where maternal swabs were negative for Group B Streptococci.

MATERNAL SWAB POSITIVITY FOR GROUP B STREPTOCOCCI AND NEONATAL SEPSIS

Group B Streptococci in Maternal Swab	Neonatal Sepsis present	Neonatal Sepsis Absent	Total
Present	1 (0.5%)	3 (1.5%)	4 (2.0%)
Absent	4 (2.0%)	192 (96.0%)	196 (98.0%)
Total	5(2.5%)	195 (97.5%)	200 (100.0%)

Table 13 - MATERNAL SWAB POSITIVITY AND NEONATAL SEPSIS

Overall, Neonatal Sepsis was found in 2.5% of the total cases of the study.

Neonatal Sepsis was present in 20% percent of cases where maternal swabs were positive for Group B Streptococci and in 1.5% percent of cases where maternal swabs were negative for Group B Streptococci.

MATERNAL SWAB POSITIVITY FOR GROUP B STREPTOCOCCI AND PREMATURE RUPTURE OF MEMBRANES

Group B Streptococci in Maternal Swab	Premature Rupture of Membranes Present	Premature Rupture of Membranes Absent	Total
Present	1 (0.5%)	3 (1.5%)	4 (2.0%)
Absent	20 (10.0%)	176 (88.0%)	196 (98.0%)
Total	21 (10.5%)	179 (89.5%)	200 (100.0%)

Table 14 - MATERNAL SWAB POSITIVITY AND PREMATURE RUPTURE OF MEMBRANES

The overall rate of Premature Rupture of Membranes in the study patients was 21%. Premature Rupture of membranes was present in 25% percent of cases where maternal swabs were positive for group B Streptococci and in

10.2% percent of cases where maternal swabs were negative for Group B Streptococci.

NEONATAL SWAB RESULTS AND OUTCOME

One Swab taken from a neonate was found to be positive for Group B Streptococci. The mother of this neonate was also positive by vaginal swab for Group B Streptococci, the pregnancy was associated with Premature Rupture of Membranes and the neonate was diagnosed with neonatal sepsis. After treatment, the neonate made a full recovery.

PREVALENCE OF GROUP B STREPTOCOCCI AND ITS ASSOCIATION WITH MATERNAL AND NEONATAL COMPLICATIONS.

1. Prevalence of Group B *Streptococci* in Maternal Samples - 2 %
2. Incidence of Preterm Labour among the Study patients - 3.5 %
3. Association of Group B *Streptococci* in Maternal Samples with Preterm Labour - 25 %
4. Relative Risk of Group B *Streptococci* Causing Preterm Labour - 8.33
5. Incidence of Premature Rupture of Membranes Among the Study patients - 10.2 %
6. Association of Group B *Streptococci* in Maternal Samples with Premature Rupture of Membranes – 25 %
7. Relative Risk of Group B *Streptococci* Causing Premature Rupture of Membranes - 2.083
8. Incidence of Neonatal Sepsis among the Study patients - 1.5 %
9. Association of Group B *Streptococci* in

Maternal Samples with Neonatal Sepsis - 20 %

10. Relative Risk of Group B *Streptococci* Causing Neonatal Sepsis - 12.25

DISCUSSION

The Group B *Streptococci* (GBS) are known to cause a wide variety of infections in adults, but clinical interest in these bacteria mainly relates to their ability to cause serious neonatal illness, especially meningitis and sepsis. In developed countries these organisms are the leading cause of neonatal sepsis and meningitis with a case fatality rate of 40 to 80%. [85]. The reason for this is not clear. However, in developing countries like India, the problem has not been adequately studied and there are only a few reports available [86,87,88,89]. In the present study most of the females were from lower socioeconomic group and in 18 to 35 years of age group.

In our study of 200 women, we have found a prevalence of GBS colonization in mothers to be 2.0%. This is comparable with other studies conducted in India, such as those by Kulkarni et al, Mhaskarrita et al, and Singh et al, which have found a prevalence ranging from 1.3% to 2.5%. [42, 43, 44, 45, 46, 47]

Studies conducted in other countries, however have prevalance rates to be higher, ranging from 6.4% to 35.0% [28, 29, 30, 31, 32, 33, 34, 38]. The reason for this difference between studies conducted in India and other countries is not clear.

In adult women, GBS carriage in the genital tract, perineal skin and gastrointestinal tract is of great importance in view of its significance in GBS neonatal infection, whether asymptomatic mucous membrane colonization or

symptomatic invasive infection (early onset septicemia, meningitis etc.) In the present study we found that only one neonate was colonized by GBS. Thus an invasive attack rate was found to low. This might be due to passive immunization of newborns via transplacental transfer of type specific serum antibodies from their mothers. Also there was complete absence of GBS colonization in neonates of non colonized mothers, and this probably indicates that nosocomial infection of GBS was not a major problem in this hospital.

Considerable work has been done to determine the complications associated with GBS carriage. A number of factors are involved, like maternal urinary tract infection, premature or prolonged rupture of membranes, premature delivery, and peak intrapartum fever greater than $37.5^{O}C$.

Some workers give weightage to lack of type specific antibodies by neonates due to failure of transplacental transfer of these antibodies. This is responsible for GBS colonization, or in turn GBS disease in the newborn.

For better isolation of GBS from clinical material the use of transport medium has revealed the critical consideration. Several works tried different methods of transportation of clinical specimens from patients to the laboratory. In the present study we used Todd-Hewitt's broth as selective broth, and the samples were then cultured in Sheep Blood Agar.

SEROTYPES AND FACTORS AFFECTING VIRULENCE

The Group B β-hemolytic *Streptococcus* (*S. agalactiae*) contains a Lancefield-grouping antigen, a type-specific cell-surface polysaccharide and protein antigens.

The prevalence of the various Group B capsular serotypes varies over time and may differ from place to place. Prior to the 1990s, most Group B *Streptococcal* disease was caused by serotypes Ia, Ib, II, III, and V; serotypes IV and VI through VIII were relatively uncommon. During the early to mid-1990s, serotype V stains began to emerge, with the percentage of isolates in this group increasing from 2.6% in 1992 to 20% in 1994 [90,91]. Studies conducted in the U.S. and abroad indicate that serotypes Ia, Ib, II, III and V now predominate among vaginal isolates and clinical isolates from patients [92,94,94]. Recently, serotypes VI and VII have appeared as the predominant serotypes in Japan [95].

Neonatal early-onset disease due to serotype VIII has also been reported in Japan [96]. Type II strains of GBS account for 60% of isolates from cases of neonates sepsis and over 80% of isolates from infants with meningitis, suggesting that this GBS serotype possesses enhanced virulence [97]. The type III capsular polysaccharide is composed of a repeating structural backbone consisting of galactose, glucose, and N-acetyl-neuraminic acid (sialic acid). The presence of this molecule on the surface of the organism inhibits activation of

the alternative complement cascade and prevents phagocytosis. Removal of sialic acid residues with neuraminidase leads to complement activation phagocytosis, and intracellular killing of the organisms and diminished virulence on intravenous challenge in a rat model [98,99].

GBS also produce a variety of other potential virulence determinants. Like the Group A *Streptococci*,GBS also produce C5a peptidase. C5a is a complement component cleavage product that is produced by alveolar epithelial cells, acts as an attractant for inflammatory cells, and is involved in the process of pulmonary inflammation [100]. The C5a peptidase produced by the *Streptococci* cleaves C5a at the C-terminus, thereby interfering with C5-mediated neutrophil chemotaxis [101]. New information indicates that this peptidase also binds to fibronectin [102].

Group B β-hemolytic *Streptococci* are a major cause of disease in the neonatal and perinatal periods. Women become colonize with the organism in the vagina and the rectum, and vaginal colonization is found in 10-35% of pregnant women; up to 60% of the colonized women will carry the organism intermittently [103,104]. Colonization of the vagina may actively reflect contamination from the rectum, with the gastrointestinal tract being the principal reservoir of the organism.

COMPLICATIONS

GBS is associated with a spectrum of maternal and fetal infections ranging from asymptomatic colonization to sepsis. *Streptococcus* agalactiae has been implicated in adverse pregnancy out comes, including preterm labor, prematurely ruptured membranes, clinical and subclinical chorioamnionitis, and fetal and neonatal infections. It is associated with about 20% of postpartum endometritis, 25% of bactremias following caesarean section, and 25-30% of cases of asymptomatic bacteriuria during and after pregnancy, they are also associated with a variety of infections in nonpregnant adults [105].

NEONATAL INFECTIONS

Early-Onset disease occurs with an incidence of 0.7 in 1.000 to 3.7 in 1,000 live births and is associated with in utero or perinatal organism acquisition 106. The organism is acquired either by ascending infection in utero before delivery, through ruptured fetal membranes, or during passage through a birth canal that is colonized with GBS. Although a substantial proportion of these infants (approximately 50%) will be colonized with GBS, only 1-2% of them become infected [107]. Onset of disease occurs during the first 5 days of life; in more than half the cases, infants become ill within the first 12 to 20 hours after birth [108]. The disease spectrum includes bacteremia, pneumonia,

meningitias, septic shock, and neutropenia. Although more than 50% of cases occur in full-term infants, a higher attack rate and greater morbidity are associated with preterm infants. Mortality owing to early – onset disease in full-term infants ranges from 2% to 8%; higher mortality rates are seen in premature infants and are inversely proportional to the birth weight of the neonate [109]. Maternal factors that increase the risk for early –onset infection of the neonate include premature labour, prolonged rupture of the fetal membranes, postpartum bacteremia, maternal amnionitis, heavy vaginal colonization with Group B *Streptococci*, and Group B *Streptococci* bacteriuria [110,111].

Late –onset disease occurs with an incidence of 0.5 in 1,000 to 1.8 in 1,000 live births [98]. Disease becomes clinically evident 7 days to 3 months (average, 3 to 4 weeks) after birth. Whereas about half of the late –onset infections are acquired from the birth canal of colonized mothers, the remaining cases result from postnatal organism acquisition from the mother or other caregivers or nosocomially [112]. Bacteremia with accompanying meningitis is the predominant clinical presentation [113]. Mortality associated with late-onset disease is about 10-15%. Up to 50% of children with late onset meningitis will have permanent neurologic complications and sequelae [114]. The distribution of Group B *Streptococci* serotypes also varies according to whether it is early-onset disease without meningitis, the serotype distribution is equally divided

among types Ic, II, and III. Among similarly infected neonates with meningitis, serotype III strains account for over 90% of the isolates. On the other hand, group B streptococcal meningitis in adults is associated primarily with serotype II organisms.

With the decline in neonatal Group B *Streptococcal* disease, over two-thirds of infections in adults are not associated with pregnancy. Adults with group B disease usually have significant underlying disease, including diabetes, liver cirrhosis, stroke, neoplasia, or urinary tract dysfunction [115]. Skin and soft-tissue infections are the most common clinical entities associated with invasive Group B *Streptococci* and include cellutitis, abscesses, infected decubitus ulcers, and invasive would infections following surgical procedures [116]. Ostemyelitis may occur as a complication of cellulites by contiguous spread, particularly in association with decubitus ulcers, or as a result of hematogenous seeding from another site of infection. Group B *streptococci* are also the major cause of osteomyelitis in infants, with hematogenous seeding of bone being the source of the organism.

Bacteruria with Group B *Streptococci* has been associated with adverse pregnancy outcome, increased rates of premature labor, and premature rupture of fetal membranes. Besides being a well-recognized cause of urinary tract infection in pregnant women, this organism is also a cause of cystitis and

pyelonephritis in men, nonpregnant women, and children. Anywhere from 5% to over 20% of nonpregnant adults with bacteremia will have Group B *streptococcal* urinary tract infection [117]. Risk factors for urinary tract infection caused by Group B *Streptococci* include advanced age, underlying disease (especially diabetes) , presence of an indwelling urinary catheter, prior urinary tract infections, structural abnormalities of the urinary. Pyelonephritis and renal abscess are potential complications of both ascending infection and hematogenous dissemination of Group B *Streptococci* [118].

By the mid-1980s, increased knowledge regarding group B *Streptococci* infections and the recognition of the symptoms in at-risk patients led to improvements in neonatal care that resulted in reduction of the fatality rate to about 15%. Since infants born to heavily colonized mothers are more likely to have early-onset disease and because infants who acquire a large bacterial inoculum during birth have significantly increased likelihoods of having both early-and late- onset disease, the identification of colonized mothers became a central focus for prevention strategies. Investigators examined possible interventions to prevent GBS disease, and several clinical trials demonstrated that intrapartum administration of antimicrobial agents interrupted the transmission of GBS*i* from the mother to the neonate and reduced the incidence of early-onset infections [119,120,121]. This chemoprophylactic approach prevented

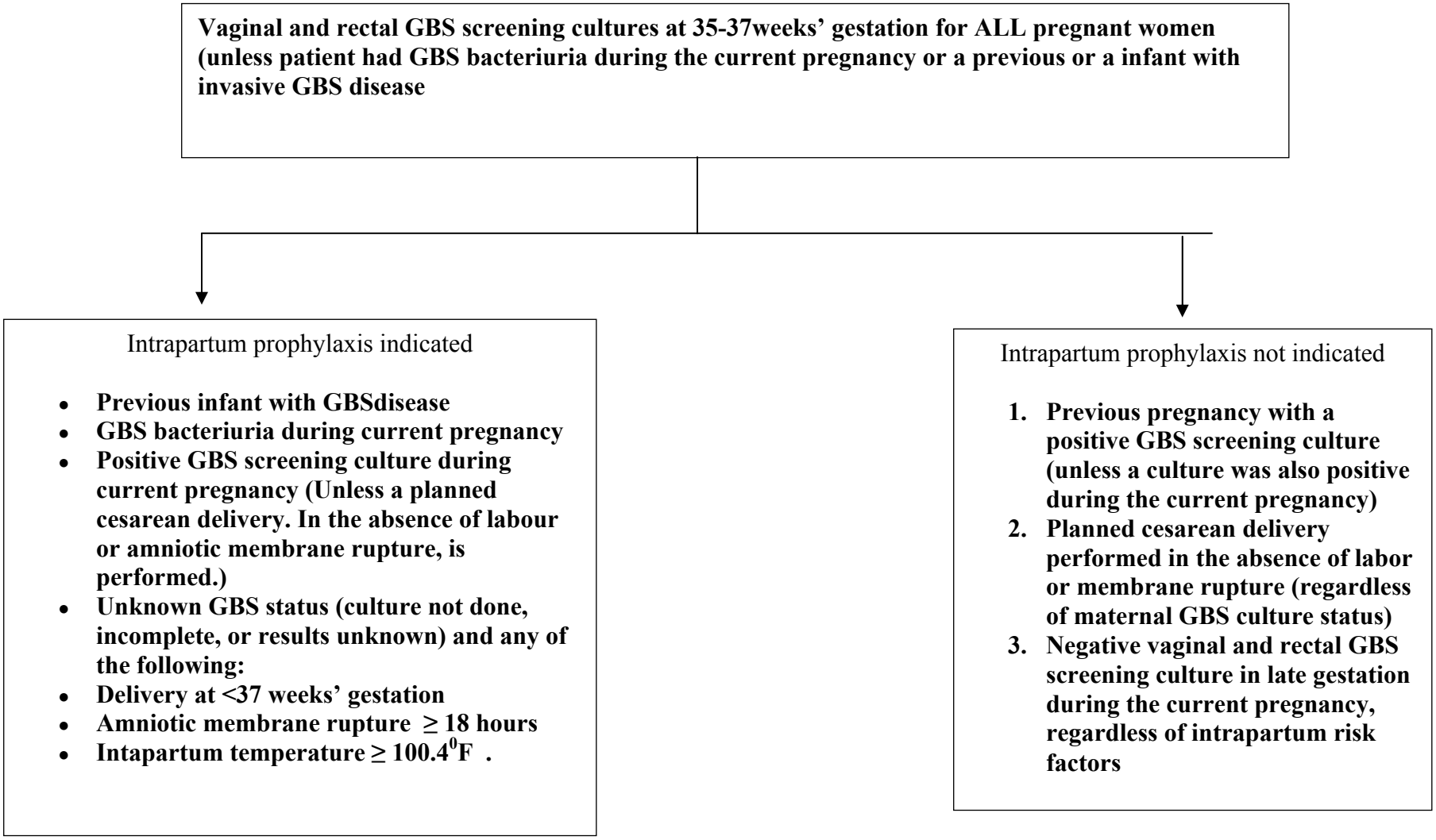
about 70 -75% of early-onset disease, but it had no effect on the development of late-onset disease. Several manufactures of microbiology products started to develop direct detection methods for Group B *Streptococci* in vaginal swab specimens similar to those used for direct detection of Group A *Streptococci* in throat swab specimens. These tests used anti- Group B *Streptococci* antibodies to detect organisms directly by latex agglutination or rapid visual or colormetric immunoassay formats. Theoretically, these rapid test could be performed in the immediate prepartum period to determine colonization status, and antimicrobial chemoprophylaxis could be administered prior to and during delivery if the antepartum assays were positive. These commercial rapid tests for direct detection of Group B *Streptococci* in vaginal swab specimens varied significantly in sensitivity (i.e., 11-88%) when compared with overnight broth techniques and, in general, identified only women who were heavily colonized [122, 123,124].

RECOMMENDED PREVENTION STRATEGIES

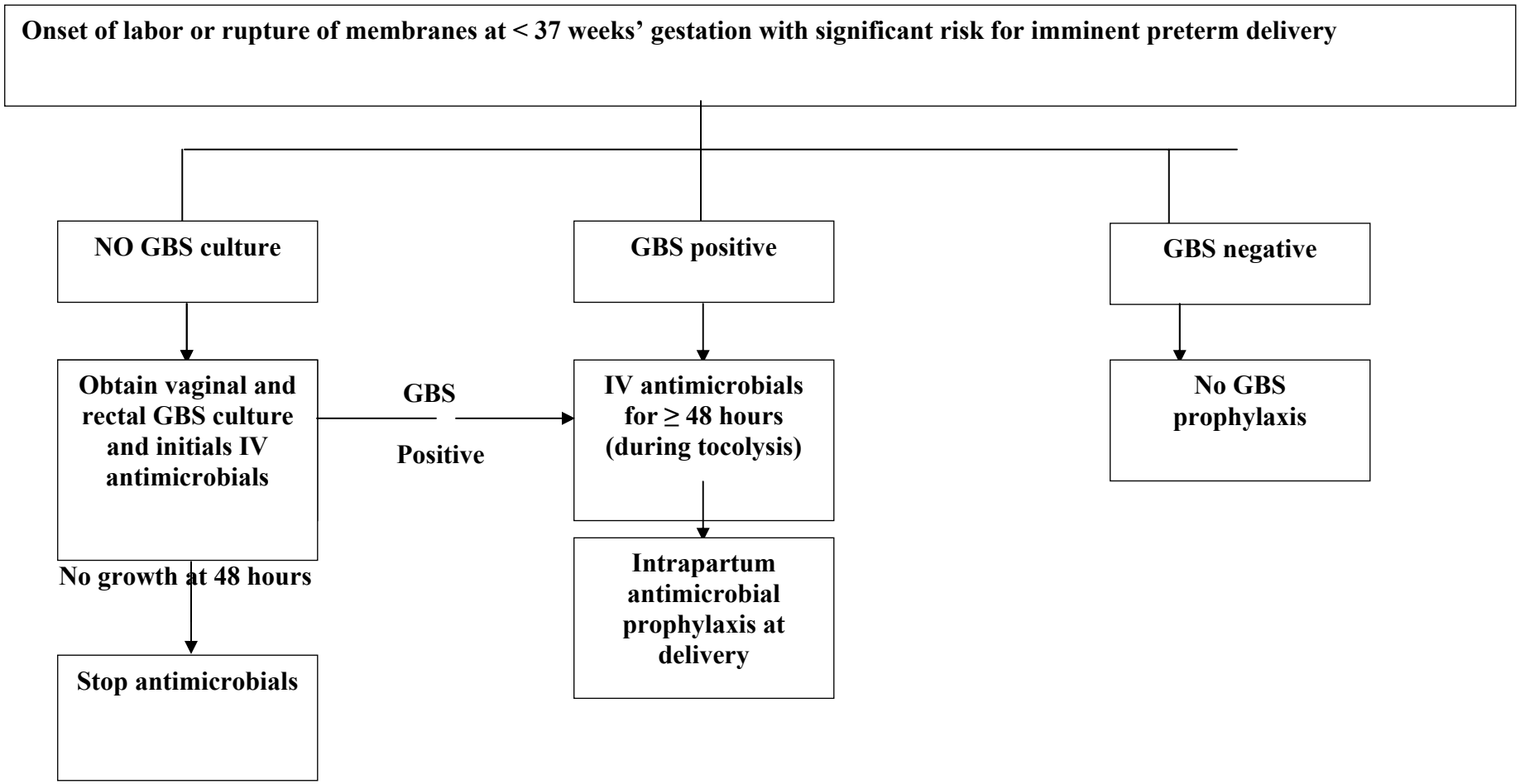
In 1996, the Centers for Disease Control and Prevention (CDC), together with the American Academy of Pediatrics (AAP) and the American College of Obstetrics and Gynecology (ACOG) issued consensus recommendations on a prevention strategy for Group B *Streptococcal* disease [125,126,127].

These guidelines recommended that obstetrical healthcare providers adopt either a culture –based or a risk-based strategy for the prevention of early-onset Group B *Streptococcal* disease. A critical component of these guidelines is the use of maternal antimicrobial prophylaxis during labor and delivery. To date, no penicillin-resistant strains of Group B *Streptococci* have been isolated, although clindamycin and erythromycin resistance has become relatively common.

In 2002, the Committee on Obstetrical Practice issued a statement supporting the use of culture-based prevention strategies based on data from the Active Bacterial Core Surveillance Emerging Infections Program network suggesting that the culture-based approach was superior to the risk-based approach [129]. New guidelines were issued by the CDC in 2002, replacing the 1996 guidelines and recommending universal, pre-natal, culture-based screening for vaginal and rectal colonization of all pregnant women at 35-37 weeks of gestation.



CDC GUIDELINES FOR GROUP B STREPTOCOCCUS PROPHYLAXIS



CDC GUIDELINES FOR GROUP B STREPTOCOCCUS PROPHYLAXIS (CONT'D)

In our study one Swab taken from a neonate was found to be positive for Group B *Streptococci.* The mother of this neonate was also positive by vaginal swab for GBS, the pregnancy was associated with Premature Rupture of Membranes and the neonate was diagnosed with neonatal sepsis. After treatment, the neonate made a full recovery.

It was also found that the incidence of Preterm Labour in those mothers who were positive for GBS was 25.0%; compared to 3.5% in those mothers who were negative for GBS.

Similarly, the incidence of Premature Rupture of Membranes in those mothers who were positive for GBS was 25.0%; compared to 10.2% in those mothers who were negative for GBS.

Like the Group A *streptococci,* isolates of GBS remain suseptiple to penicillin G, the drug of choice for treatment of infections and for perinatal chemoprophylaxis of women who are vaginal carriers of Group B *streptococci*. Ampicillin, cefotaxime, cefotaxone, cefezolin, and meropenem are also highly active. Some GBS demonstrate resistance to erythromycin and clindamycin.

In our study GBS was sensitive to Penicillin, Vancomycin, Erythromycin , Ciprofloxacin and Tetracyclin, but was resistant to Gentamycin and Amikacin .

The incidence of Neonatal Sepsis in neonates born to those mothers who were positive for GBS was 20.0%; compared to 1.5% in those mothers who were negative for GBS. This indicates a maternal to fetal transmission rate of 20.0% which is less than the 50 % maternal to fetal transmission rate reported in other studies.

These results indicate that there is a definitive increased risk of Pre-term Labour, Premature rupture of Membranes and Neonatal Sepsis in pregnant women who are colonized with GBS.

CONCLUSIONS

Group B *Streptococcus* colonization in the pregnant mother has been associated with a number of maternal and neonatal complications. These include higher rates of Premature Rupture of Membranes, Premature Delivery, Puerperal Infection, and Neonatal Sepsis.

In our study we have attempted to determine the prevalence of colonization of Group B *Streptococci* in mothers at the time of admission to the Labour Room and in neonates soon after delivery. The study patients were then followed up in order to detect any association with maternal and nonatal complications. Our study excluded Caesarean deliveries.

In our study, the prevalence of Group B *Streptococci* in the Maternal samples was found to be 2.0%. The incidence of Preterm Labour in those mothers who were positive for Group B *Streptococcus* was 25.0%; compared to 3.5% in those mothers who were negative for Group B *Streptococcus.*

Similarly, the incidence of Premature Rupture of Membranes in those mothers who were positive for Group B *Streptococcus* was 25.0%; compared to 10.2% in those mothers who were negative for Group B *Streptococcus.*

The incidence of Neonatal Sepsis in neonates born to those mothers who were positive for Group B *Streptococcus* was 20.0%; compared to 1.5% in those mothers who were negative for Group B *Streptococcus.*

These results indicate that there is a definite increased risk of Pre-term Labour, Premature rupture of Membranes and Neonatal Sepsis in pregnant women who are colonized with Group B *Streptococcus.*

It was also found that the samples of Group B *Streptococci* obtained in our study was sensitive to Vancomycin,Erythromycin,Penicillin,Ciprofloxacin and tetracycline and showed resistance to Amikacin and Gentamycin.

The prevalence of maternal colonization with Group B *Streptococci* in our study population is low at only 2 %, which is comparable with other studies conducted in India (1.3 – 2.5 %). But the reported prevalence in Western countries is higher ranging from 6.4 – 35 %. The association of pre-term labour, pre-mature rupture of membranes and neonatal sepsis was found significant in pregnant women who were colonized with Group B *Streptococci.* As the prevalence of Group B *Streptococci* is low, it is not cost effective to do a culture based screening at 37 weeks of gestation for all pregnant women as per ACOG and CDC guidelines.

The low occurrence of Group B Streptococci maternal colonization (2%) and neonatal transmission (0.5 %) shows that selective risk factor based screening and anti-biotic prophylaxis should be considered as an effective protocol for preventing neonatal morbidity and mortality due to Group B Streptococci than a mass screening for Group B Streptococci during pregnancy.

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PROFORMA

NAME : ADDRESS :

AGE :

PARITY : SOCIO ECONOMIC STATUS:

GESTATIONAL AGE AT ONSET OF LABOUR :

OBSTETRIC HISTORY :

PAST HISTORY :

FAMILY HISTORY:

DETAILS OF PRESENT PREGNANCY :

UTI IN CURRENT PREGNANCY :

GENERAL EXAMINATION:

VAGINAL EXAMINATION FINDINGS :

SWAB : VAGINAL INTROITUS TO ANUS :

TIME OF RUPTURE OF MEMBRANES :

DURATION OF LABOUR :

MADE OF DELIVERY :

SEX :

WEIGHT OF BABY :

REPORT OF VAGINAL SWAB CULTURE :
SWABS FROM NEONATE :

SITES	REPORTS

1) NASAL SWAB
2) THROAT SWAB
3) UMBILICAL SWAB
4) EXTERNAL EAR SWAB

POST PARTTUM COMPLICATIONS:

1) FEVER
2) UTI
3) FOUR SMELLING LOCHIA

* * *

S.No	IP Number	age	obstetrical score	Socio economic class	GA at onset of labour	PROM	Sex of Baby	Weight of Baby	Report swab for GBS		Post Partum Complications		
			Primigravida Multigravida						Maternal	Neonatal	Fever	UTI	Lochia foul smelling
1	704279	34	M	II	Term	-	M	3.5	-	-	-	-	-
2	704264	25	P	III	Term	-	F	3	-	-	-	-	-
3	702361	21	P	IV	Term	-	M	2.4	-	-	-	-	-
4	702843	24	M	III	Term	-	F	2.75	-	-	-	-	-
5	712803	31	M	V	Term	-	M	2.65	-	-	-	-	-
6	703108	26	P	V	Term	-	M	2.75	+	-	-	-	-
7	703430	21	M	III	Term	-	F	2.8	-	-	-	-	-
8	703545	22	P	V	36	+	M	2.3	-	-	-	-	-
9	703378	18	P	I	Term	-	F	3.15	-	-	-	-	-
10	703378	24	M	IV	Term	-	M	3	-	-	-	-	-
11	703782	23	P	V	Term	-	F	2.65	-	-	-	-	-
12	703962	23	M	III	Term	-	M	2.95	-	-	-	-	-
13	704240	27	P	V	Term	-	F	2.5	-	-	-	-	-
14	704149	24	M	IV	Term	-	M	2.7	-	-	-	-	-
15	704433	23	P	II	Term	-	M	2.8	-	-	-	-	-
16	704450	32	P	V	Term	-	F	3.75	-	-	-	-	-
17	704533	21	M	III	Term	+	M	2.8	-	-	-	-	-
18	703632	17	P	V	Term	-	M	2.9	-	-	-	-	-
19	702985	23	M	II	Term	-	F	2.8	-	-	-	-	-
20	703025	28	P	IV	Term	-	M	2.45	-	-	-	+	-
21	703169	25	P	III	Term	-	M	2.6	-	-	-	-	-
22	704609	22	M	V	35	-	F	2.45	-	-	-	-	-
23	704722	31	P	IV	Term	-	M	2.8	-	-	-	-	-
24	704870	24	P	II	Term	-	F	2.3	-	-	-	-	-
25	704671	23	M	V	Term	+	M	2.8	-	-	-	-	-

26	704652	27	M	IV	Term	-	F	2.8	-	-	-	-	-
27	704772	28	P	II	Term	-	M	3.55	-	-	-	-	-
28	704789	22	P	V	Term	-	F	3.4	-	-	-	-	-
29	704670	21	M	V	Term	-	M	3.1	-	-	-	-	-
30	704897	23	P	III	Term	-	F	3.2	-	-	-	-	-
31	704924	18	P	IV	Term	+	M	2.3	-	-	-	-	-
32	704988	21	M	II	Term	-	M	3.1	-	-	-	-	-
33	705171	25	P	IV	Term	-	F	3	-	-	-	+	-
34	705199	21	M	III	36	-	M	2.5	-	-	-	-	-
35	705362	32	P	IV	Term	-	F	2.8	-	-	-	-	-
36	705395	22	P	III	Term	-	M	2.75	-	-	-	-	-
37	705180	24	M	II	Term	+	M	2.65	-	-	-	-	-
38	705047	23	M	V	Term	-	M	2.5	-	-	-	-	-
39	704714	30	P	V	Term	-	M	3.6	-	-	-	-	-
40	701026	24	P	V	Term	-	M	2.8	-	-	-	-	-
41	704517	31	M	V	Term	-	F	3.1	-	-	-	-	-
42	5017135	27	P	II	Term	-	M	3	-	-	-	-	-
43	603550	18	P	V	Term	-	F	2.9	-	-	-	-	-
44	603283	23	M	III	Term	-	M	2.4	-	-	-	-	-
45	603092	25	P	IV	Term	-	M	2.95	-	-	-	-	-
46	602731	28	M	IV	Term	-	F	2.9	-	-	-	-	-
47	602524	29	P	V	Term	-	M	2.9	-	-	-	-	-
48	601821	24	P	III	Term	-	M	2.7	-	-	-	-	-
49	601832	23	M	IV	Term	-	F	2.3	-	-	-	-	-
50	601569	32	P	V	Term	+	M	2.8	-	-	-	-	-
51	601444	25	P	IV	Term	-	F	3	-	-	-	-	-
52	601589	26	M	V	Term	-	M	3.2	-	-	-	+	-
53	601376	24	M	V	Term	-	F	3.2	-	-	-	-	-
54	600963	23	P	III	Term	-	M	3.1	-	-	-	-	-
55	601027	21	P	V	Term	-	F	3	-	-	-	-	-

56	600725	18	M	IV	Term	-	M	3	-	-	-	-	-
57	601024	30	P	II	32	-	M	2.25	-	-	-	-	-
58	600877	21	M	III	Term	-	F	3.2	-	-	-	-	-
59	600740	23	P	IV	Term	-	F	3.15	-	-	-	-	-
60	600705	23	P	IV	Term	-	M	2.85	-	-	-	-	-
61	600594	19	M	I	Term	-	F	2.65	-	-	-	-	-
62	600880	26	M	III	Term	-	M	2.8	-	-	-	-	-
63	600828	25	M	IV	Term	-	F	2.9	-	-	-	-	-
64	600741	33	P	V	Term	-	M	3.2	-	-	-	-	-
65	600468	24	P	IV	Term	+	M	2.25	-	-	-	+	-
66	600566	25	M	V	Term	-	F	3	-	-	-	-	-
67	600204	24	M	III	Term	-	M	2.8	-	-	-	-	-
68	603456	31	P	IV	Term	-	F	3.2	-	-	-	-	-
69	502324	26	P	II	Term	-	M	3	-	-	-	-	-
70	502331	23	M	III	Term	-	M	3.1	-	-	-	-	-
71	503211	25	P	V	Term	-	M	3.1	-	-	-	-	-
72	502297	31	P	III	Term	-	F	2.5	-	-	-	-	-
73	502298	27	M	V	Term	+	M	3.2	-	-	-	-	-
74	502281	24	P	II	Term	-	M	3.1	-	-	-	-	-
75	502274	23	P	IV	Term	-	F	3	-	-	-	-	-
76	502201	26	P	V	35	-	M	2.4	+	-	-	-	+
77	502160	25	M	III	Term	-	F	3.2	-	-	-	-	-
78	502147	26	P	IV	Term	-	M	3	-	-	-	-	-
79	502080	24	M	V	Term	-	F	3.1	-	-	-	-	-
80	502026	23	P	V	Term	-	M	3	-	-	-	-	-
81	501992	30	M	IV	Term	-	F	2.2	-	-	-	-	-
82	501992	25	P	II	Term	-	F	2.45	-	-	-	+	-
83	501965	23	P	V	Term	-	M	2.7	-	-	-	-	-
84	501949	17	P	V	Term	-	F	2.8	-	-	-	-	-
85	501871	22	P	IV	Term	-	M	2.9	-	-	-	-	-

86	501877	21	M	III	Term	-	F	3.2	-	-	-	-	-
87	501873	22	M	V	Term	+	M	3	-	-	-	-	-
88	501774	23	P	IV	Term	-	F	3.1	-	-	-	-	-
89	501878	23	P	III	Term	-	M	2.3	-	-	-	-	-
90	501763	35	P	V	Term	-	F	3	-	-	-	-	-
91	501747	25	M	II	Term	-	M	2.9	-	-	-	-	-
92	501749	24	M	III	Term	-	F	2.8	-	-	-	-	-
93	501744	23	P	V	Term	-	F	2.3	-	-	-	-	-
94	501750	23	M	IV	Term	-	F	2.8	-	-	-	-	-
95	502005	24	P	V	Term	-	M	3.2	-	-	-	-	-
96	701452	21	M	III	Term	-	M	2.4	-	-	-	+	-
97	603616	32	P	V	Term	-	F	3.1	-	-	-	-	-
98	601605	23	P	II	Term	-	M	3	-	-	-	-	-
99	601199	24	M	V	Term	-	F	2.9	-	-	-	-	-
100	601121	25	P	V	Term	-	M	2.5	-	-	-	-	-
101	601302	24	M	II	Term	-	F	2.8	-	-	-	-	-
102	601122	24	P	IV	Term	-	M	2.8	-	-	-	-	-
103	608704	32	M	III	Term	+	M	2.4	-	-	-	-	-
104	608444	25	P	IV	Term	-	M	3	-	-	-	-	-
105	608380	24	M	V	Term	-	F	2.9	-	-	-	-	-
106	607762	25	M	III	Term	-	M	2.4	-	-	-	-	-
107	609826	23	M	IV	Term	-	F	3.2	-	-	-	-	-
108	601247	32	P	V	Term	-	M	3	-	-	-	-	-
109	601277	24	M	III	Term	-	F	3.1	-	-	-	-	-
110	601266	25	M	IV	Term	-	M	2.5	-	-	-	-	-
111	706372	17	P	V	Term	-	F	3	-	-	-	-	-
112	706434	24	M	V	Term	-	M	2.7	-	-	-	-	-
113	702627	26	P	IV	Term	+	F	2.9	-	-	-	+	-
114	703131	27	M	II	Term	-	M	2.8	-	-	-	-	-
115	703179	24	P	V	Term	-	M	3	-	-	-	-	-

116	703417	25	M	V	Term	-	F	3.2	-	-	-	-	-
117	703726	21	P	IV	Term	-	M	3.1	-	-	-	-	-
118	704149	23	M	V	Term	-	F	3.	-	-	-	-	-
119	702361	34	P	III	Term	-	M	2.4	-	-	-	-	-
120	704264	22	M	IV	Term	-	M	3	-	-	-	-	-
121	704279	22	P	V	Term	+	F	3.2	-	-	-	-	-
122	702354	23	M	V	Term	-	M	3	-	-	-	-	-
123	704517	21	M	III	Term	-	M	3.2	-	-	-	-	-
124	706581	23	M	V	Term	-	F	3.1	-	-	-	-	-
125	706373	24	p	IV	Term	-	M	3	-	-	-	-	-
126	706621	23	P	V	Term	-	M	2.8	-	-	-	-	-
127	701440	19	M	II	Term	-	F	2.5	-	-	-	-	-
128	706732	25	P	IV	Term	-	M	3.1	-	-	-	-	-
129	706741	25	M	V	Term	-	F	3.3	-	-	-	-	-
130	706728	36	P	V	Term	-	M	3.1	-	-	-	-	-
131	706708	27	M	V	Term	-	F	3	-	-	-	-	-
132	705593	23	M	IV	Term	+	M	2.9	-	-	-	-	-
133	705748	21	M	V	Term	-	M	2.8	-	-	-	-	-
134	705878	22	P	V	Term	+	M	2.5	-	-	-	-	-
135	705967	22	M	IV	Term	-	F	2.8	-	-	-	-	-
136	705897	21	P	V	Term	-	M	3	-	-	-	-	-
137	706038	22	M	V	Term	-	M	3.2	-	-	-	-	-
138	706134	31	M	V	Term	-	M	3.21	-	-	-	-	-
139	706181	24	M	IV	Term	-	M	3.1	-	-	-	-	-
140	705670	24	P	II	Term	+	F	3	-	-	-	-	-
141	706320	23	M	V	Term	-	M	3	-	-	-	-	-
142	706262	25	P	IV	Term	-	M	2.8	-	-	-	-	-
143	706578	26	M	V	Term	-	M	2.2	-	-	-	-	-
144	706502	24	M	II	Term	-	F	2.9	-	-	-	-	-
145	705243	25	P	V	Term	-	F	2.8	-	-	-	-	-

146	705573	23	M	IV	Term	-	M	3.6	-	-	-	-	-
147	705684	24	P	V	Term	-	M	3	-	-	-	-	-
148	705685	24	M	IV	Term	-	F	2.7	-	-	-	-	-
149	705678	25	P	V	Term	-	F	3.2	+	-	-	-	-
150	705859	21	P	V	Term	-	F	2.8	-	-	-	-	-
151	705569	21	M	IV	Term	-	M	3	-	-	-	-	-
152	705859	19	M	V	Term	-	F	2.9	-	-	-	-	-
153	705569	23	P	II	Term	-	M	2.8	-	-	-	-	-
154	706124	23	P	II	Term	-	F	2.7	-	-	-	-	-
155	706126	24	P	V	Term	-	M	3	-	-	-	-	-
156	706372	24	M	IV	36	+	M	2.4	-	-	-	-	-
157	707209	23	M	V	Term	-	F	2.9	-	-	-	-	-
158	707326	25	P	IV	Term	-	M	2.7	-	-	-	-	-
159	707293	26	M	V	Term	-	F	3	-	-	-	-	-
160	707343	24	P	V	Term	-	M	2.8	-	-	-	-	-
161	701561	33	M	II	Term	+	F	2.9	-	-	-	-	-
162	707295	21	P	V	Term	-	M	2.75	-	-	-	-	-
163	707432	23	M	IV	Term	-	F	2.8	-	-	-	-	-
164	707449	24	M	V	Term	-	M	3.2	-	-	-	-	-
165	707496	25	P	V	Term	-	F	3.75	-	-	-	-	-
166	707468	26	M	IV	Term	-	M	3	-	-	-	-	-
167	707538	18	M	V	Term	-	F	3.2	-	-	-	-	-
168	707388	35	P	II	Term	-	M	3.1	-	-	-	-	-
169	707719	21	M	IV	Term	-	F	3	-	-	-	-	-
170	707812	22	M	V	Term	-	M	2.9	-	-	-	-	-
171	706794	22	M	IV	Term	-	M	2.8	-	-	-	-	-
172	707036	33	M	II	Term	-	F	2.5	-	-	-	-	-
173	706994	24	P	V	Term	+	M	2.4	-	-	-	-	-
174	706839	21	P	IV	Term	-	F	2.9	-	-	-	-	-
175	706988	23	M	IV	Term	-	M	2.8	-	-	-	-	-

176	706989	25	P	V	Term	-	F	3	-	-	-	-	-
177	706507	26	M	IV	Term	-	F	3	-	-	-	-	-
178	708714	28	P	V	Term	+	M	2.7	+	-	-	-	-
179	708573	17	M	V	Term	-	F	3.1	-	-	-	-	-
180	708551	26	P	II	Term	-	F	3	-	-	-	-	-
181	708531	28	M	IV	Term	-	M	2.4	-	-	-	-	-
182	707928	21	P	V	Term	-	F	2.6	-	-	-	-	-
183	708238	32	M	IV	Term	-	F	2.9	-	-	-	-	-
184	708206	23	P	V	Term	-	M	2.8	-	-	-	-	-
185	708082	24	M	II	Term	-	F	2.9	-	-	-	-	-
186	707928	25	P	IV	Term	-	F	2.8	-	-	-	-	-
187	707926	21	P	V	Term	-	M	2.7	-	-	-	-	-
188	708168	22	M	V	Term	-	F	2.8	-	-	-	-	-
189	707782	30	P	IV	36	-	M	2.5	-	-	-	+	-
190	707884	24	M	V	Term	-	F	3	-	-	-	-	-
191	707977	21	M	IV	Term	-	F	3.1	-	-	-	-	-
192	707539	21	P	V	Term	+	F	3.1	-	-	-	-	-
193	707790	23	P	V	Term	-	F	3	-	-	-	-	-
194	707849	31	P	II	Term	-	F	2.6	-	-	-	-	-
195	707795	26	M	V	Term	-	F	2.9	-	-	-	-	-
196	707641	23	P	IV	Term	-	F	2.6	-	-	-	-	-
197	777650	25	M	IV	Term	-	F	2.7	-	-	-	-	-
198	707624	24	P	V	Term	-	F	3.2	-	-	-	-	-
199	707705	21	M	V	Term	-	M	2.45	-	-	-	-	-
200	707705	20	P	V	Term	-	F	3	-	-	-	-	-